CLINICAL PROFILE AND OUTCOME OF COMMUNITY ACQUIRED ACUTE KIDNEY INJURY

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Dr. Sourabh Gupta.

ABBREVIATIONS

ACEI -	Angiotensin converting enzyme inhibitors
ACN -	Acute cortical necrosis
ADQI -	Acute dialysis quality initiative
AIN -	Acute intertial nephritis
AKI -	Acute kidney injury
AKIN -	Acute Kidney Injury Network
ANCA -	Anti neutrophil cytoplasmic antibody
APH -	Antepartum haemorrhage
ARB -	Angiotensin receptor blocker
ARF -	Acute renal failure
ATN -	Acute tubular necrosis
BPH -	Benign prostatic hypertrophy
BUN -	Blood urea nitrogen
C.R.-	Complete recovery
CA-AKI -	Community acquired-AKI
CAD -	Coronary artery disease
CAM -	Complementary and alternative medicine
CKD -	Chronic kidney disease
CLD -	Chronic liver disease
CNSD -	Central nervous system disease
COAD -	Chronic obstructive airway disease
CRRT -	Continuous renal replacement therapy
CVA -	Cerebro-vascular accident
CVSD -	Cardiovascular disease
CVVHDF -	Continuous veno-venous hemodiafiltration
DM -	Diabetes mellitus
G6PD -	Glucose 6 phosphate dehydrogenase
GBM -	Glomerular basement membrane

GFR -	Glomerular filtration rate
HA-AKI -	Hospital acquired-AKI
HBV -	Hepatitis B virus
HCV -	Hepatitis C virus
HD -	Hemodialysis
HELLP-	Hemolysis, elevated liver enzymes, low platelets
HIV -	Human immunodeficiency virus
HRS -	Hepatorenal syndrome
HTN -	Hypertension
HUS -	Hemolytic uremic syndrome
ICU -	Intensive care unit
IgA -	Immunoglobulin A
IRGN -	Infection related glomerulonephritis
ISN -	International society of nephrology
IUD -	Intrauterine death
KDIGO -	Kidney disease - improving global outcome
LLMIC -	Low & low middle income countries
MDRD -	Modification of diet in renal disease
MED -	Medical
MM -	Multiple myeloma
MPGN -	Membranoproliferative glomerulonephritis
N.R.-	Non-recovery
NK -	Not known
NSAIDS -	Non-steroidal anti-inflammatory drugs
OBS -	Obstetric
P.R -	Partial recovery
PD -	Peritoneal dialysis
PPH -	Postpartum haemorrhage
RA -	Rheumatoid arthritis
RIFLE -	Risk, Injury, Failure, Loss of kidney function, and end-stage kidney disease

RRT -	Renal replacement therapy
RSD -	Respiratory system disease
SBP -	Spontaneous bacterial peritonitis
SCr -	Serum creatinine
SLE -	Systemic lupus erythematosus
SLED -	Sustained low efficiency dialysis
SOFA -	Sequential Organ Failure Assessment
SUR -	Surgical
TB -	Tuberculosis
TLS -	Tumour lysis syndrome
TMA -	Thrombotic microangiopathy
U.O.-	Urinary obstruction

ABSTRACT

Introduction: Community acquired-AKI (CA-AKI) though well-known but is less studied. Uncovering the local features is necessary for formulating site-specific guidelines.

Aim: To identify the various etiologies, high risk group and outcome of CA-AKI.

Methods: Prospective observational study done from March 2018 to June 2019. Patients with age $\geq$ 18 years with AKI at or within 48 of admission were included. Patients hospitalized outside for 48 hours, on maintenance dialysis, post renal transplant patients or with age $\geq$ 70 years were excluded.

Results: 380 patients were diagnosed to have CA-AKI. 57% patients had pre-existing co-morbidity. Medical causes contributed to 76 % of cases, surgical 17% and obstetric 6%. Sepsis was the most common cause and had the highest mortality. 2nd and 3rd common causes were hypovolemia and tropical infections with dengue being most common. Drugs, toxins and snake bite contributed to significant cases.11 % cases were due to urinary obstruction. Obstetric cases were few still puerperal sepsis being the common obstetric cause. Majority of patient had AKI stage 3 highlighting delayed presentation. 41% required ICU stay, and 32% renal replacement therapy. Haemodialysis was the most common initial modality (40%) followed by SLED (30%), Peritoneal dialysis (25%) and CVVHDF (5%). 7 day and 30-day mortality was 11% and 19% with 30% readmission rate. 9% had no recovery with 5% remaining HD dependent, 27% had partial recovery only 45% had complete recovery. In multivariate analysis, Factors affecting early mortality were sepsis, multiple comorbidities, need of inotropes and dialysis. Overall mortality was affected by underlying CKD, CLD, multiple comorbidities, sepsis, poisoning, need of inotropes and dialysis.

Conclusion: Unmet needs in the area of CA-AKI are high. Preventable causes of AKI are still common requiring better public health initiatives. Sepsis is a common cause in patient with comorbidities. With 19% mortality and only 45% complete recovery it highlights need of better preventive strategies.

Keywords: community acquired AKI, sepsis, renal outcome, co-morbidities.

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INTRODUCTION

Acute kidney injury (AKI), even when mild, is a well-known contributor to increased mortality and morbidity in hospitalized patients.[1] Hospital acquired-AKI (HA-AKI) has been the focus of research over the last two decades. A definition that maximally utilizes the available clinical information (serum creatinine and urine output) has been developed in this setting. The most recent definition for AKI has been organized by the Kidney Disease-Improving Global Outcomes (KDIGO) initiative which is derived mainly from the data of Hospital acquired-AKI (HA-AKI).[2] Community acquired-AKI (CA-AKI) though well-known for long but as an entity has gained increased attention over the last 2-3 years.

In global snapshot of AKI by ISN, majority patients had developed community acquired AKI (60%), and it was about 80% in Low & Low Middle Income Countries (LLMICs), even when rural areas were ill-represented from these countries. It shows the importance of Community acquired-AKI (CA-AKI).[3,4]

International Society of nephrology has recognised acute kidney injury as a major health concern and it aims to curb down mortality associated with acute kidney injury to zero by 2025 (0 by 25 initiative).[3]

The disease burden in Low & Low Middle Income Countries (LLMICs) also the tropical countries is heavily influenced by local climatic and economic conditions. Thus, the spectrum of acute kidney injury (AKI) is fundamentally different to that described in affluent countries with temperate climates, where the disease burden is dominated by lifestyle related chronic diseases and degenerative disorders in the elderly and AKI usually occurs as part of multiple organ involvement in an already hospitalized, elderly patient or after surgical or diagnostic interventions, and iatrogenic factors have an important role. Moreover, in contrast to the multi factorial origin of hospital acquired AKI, the aetiology of, community acquired AKI can usually be traced to a single cause, typically an infection, dehydration, exposure to an environmental toxin,

envenomation by snakes or insects, or complications of out-of-hospital childbirth.[5]

These factors, associated with economic disparity, poor access to treatment, and social or cultural practices (such as the use of traditional herbal medicines and treatments) contribute to poor outcomes of patients with AKI.

Community-acquired AKI, though common, has been much less studied than Hospital acquired AKI. Because the causative factors of community-acquired AKI originate in the communities, environmental and lifestyle differences potentially affect the incidence and patterns of community. Therefore, uncovering the local features of community-acquired AKI is necessary for formulating site-specific preventive and therapeutic manoeuvres. Only Limited data are available on the aetiology and outcome of acute kidney injury in Indian population especially from this part of country, which creates a lacuna in our knowledge and need for this study.

AIMS AND OBJECTIVES

- To identify the various etiologies of Community acquired - AKI.
- To identify high risk groups, who have increased risk of AKI.
- To evaluate the renal status at discharge and at 30 days post admission, 7 day and 30-day mortality, Need of ICU, Length of ICU stay and Total hospital stay and Readmission within 30 days of discharge.

REVIEW OF LITERATURE

The first description of ARF, then termed ischuria renalis, was by William Heberden in 1802. At the beginning of the twentieth century, ARF, then called Acute Bright's disease, was described in William Osler's Textbook for Medicine (1909), to be "as a consequence of toxic agents, pregnancy, burns, trauma or operations on the kidneys". During the First World War it was named "war nephritis", and was reported in several publications. It was forgotten until the Second World War, when Bywater and Beall published their classical paper on crush syndrome.

Acute tubular necrosis (ATN) was the term that was used to describe this clinical entity, because of histological evidence for patchy necrosis of renal tubules at autopsy. For many years in clinical practice, the terms ATN and ARF were used interchangeably. However, it is Homer W. Smith who is credited for the introduction of the term acute renal failure, in a chapter on acute renal failure related to traumatic injuries in his 1951 textbook "The kidney-structure and Function in Health and Disease". Until recently, a precise biochemical definition for ARF was missing. As a consequence, there was no consensus on the diagnostic criteria, resulting in multiple different definitions.[6]

A survey done in 2002 revealed at least 35 definitions in the scientific literature which led to a great disparity in the reported incidence of ARF making it difficult to compare various published studies.[7]

The concept of Acute Renal Failure (ARF) has undergone significant re-examination in recent years. Traditionally, emphasis was given to the most severe acute reduction in kidney function, as manifested by severe azotemia and often by oliguria or anuria. However, recent evidence suggests that even relatively mild injury or impairment of kidney function manifested by small changes in serum creatinine (sCr) and/or urine output (UO), is a predictor of serious clinical consequences.[1,8]

The term acute kidney injury (AKI) was coined by the Acute Dialysis Quality Initiative (ADQI) group in 2004, together with the RIFLE criteria. This was in an effort to address the lack of an agreed definition of the syndrome, which was previously termed acute renal failure and which had over 35 definitions assigned to it. The previous lack of a clear definition of acute kidney injury hampered efforts to investigate the epidemiology and full clinical and socioeconomic impact of this condition.[7,9]

In May 2002 the Acute Dialysis Quality Initiative (ADQI) group for the study of AKI, composed of nephrologists and intensivists, came together over 2 days in a conference in Vicenza (Italy), with the purpose of defining AKI. From this conference, the consensual RIFLE (Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease) classification for AKI definition emerged and was published in May 2004.

The ADQI group considered that the ideal AKI definition would have to accomplish the following criteria: easy clinical applicability, sensitivity and specificity, consider baseline SCr variations and also consider the 'acute-on-chronic' phenomenon (which means the occurrence of an acute insult over a chronically injured renal function causing its deterioration). This definition should classify AKI according to its severity and its timing of occurrence. By fulfilling these criteria, this classification should allow the detection of patients whose kidney function was slightly affected as well as patients with severe kidney function deterioration.[9,10]

The RIFLE classification is based on SCr and UO determinants, and considers three severity classes of AKI (Risk, Injury and Failure), according to the variations in SCr and/or UO, and two outcome classes (loss of kidney function and end-stage kidney disease). The patient should be classified using the criteria (SCr and/or UO) which leads to the worst classification (maximum RIFLE), for instance, if a patient was in the Risk class according to the UO but in the Injury class according to SCr variation, then the worst criteria (SCr) should be used for classifying the severity of AKI in this patient.[9,10]

According to the RIFLE criteria, acute kidney injury can be diagnosed in the presence of a fall in GFR of >25% or an increase in serum creatinine of 1.5 times from baseline over a 1-7-day period. It can also be diagnosed by a fall in urine output of <0.5ml/kg/hour in 6 hours. This diagnostic criterion incorporates the Risk severity class. Injury is defined as either a 50% fall in GFR, a doubling of serum creatinine or a fall in urine output of <0.5ml/kg/hour for 12 hours. Failure is determined by changes in the level of serum creatinine whether in the form of an increase in serum creatinine of >75% from baseline, an increase of more than three times from baseline, or a level greater than 354μmol/l (≥4 mg/dL) provided there was an increase of >44.4μmol/l from (>0.5 mg/dL) baseline. In addition, Failure is diagnosed if a patient has a fall in urine output of <0.3ml/kg/hr for 24 hours, or anuria for 12 hours. Loss is described as irreversible or persistent acute kidney injury for more than 4 weeks, and End stage renal disease if this persists for more than 3 months.[9,10]

TABLE 1: RISK, INJURY, FAILURE, LOSS OF FUNCTION AND END-STAGE DISEASE (RIFLE) CLASSIFICATION

Class	**GFR**	**UO**
Risk	↑ SCr × 1.5 or ↓ GFR >25%	<0.5 mL/kg/h × >6 h
Injury	↑ SCr × 2 or ↓ GFR >50%	<0.5 mL/kg/h × >12 h
Failure	↑ SCr × 3 or ↓ GFR >75% or if baseline SCr ≥353.6 μmol/L (≥4 mg/dL) ↑ SCr >44.2 μmol/L (>0.5 mg/dL)	<0.3 mL/kg/h × >24 h or anuria × >12 h
Loss of kidney function	Complete loss of kidney function >4 weeks	
End-stage kidney disease	Complete loss of kidney function >3 months	

In September 2005, in a meeting in Amsterdam a new classification of AKI was proposed by the Acute Kidney Injury Network (AKIN) working group composed of nephrologists, critical care physicians and other physicians specialized in AKI. The AKIN classification was published in March 2007. It modified the RIFLE classification of acute kidney injury by adding the rise in serum creatinine of 26.4µmol/l within 48 hours, because this had been shown to carry a mortality risk in various studies. In addition, acute kidney injury could be diagnosed if there was a fall in urine output of <0.5ml/kg/hour in > 6 hours, as defined by the RIFLE criteria.

In addition, a new severity staging system was added. Stage 1 incorporated the serum creatinine increase of ≥ 26.4µmol/l (≥0.3 mg/dL) from baseline, or a serum creatinine increase of a multiple of 1.5-2 from baseline, as well as the urine output requirements for the definition of acute kidney injury. Stage 2 combined the Injury and Failure criteria of RIFLE and stage 3 was similar to the Failure stage of the RIFLE criteria, but with the addition of an increase in serum creatinine of ≥44.2 µmol/L (≥0.5 mg/dL) above 354µmol (≥4 mg/dL), to incorporate the rise associated with increased risk of mortality.[9-11]

TABLE 2: THE AKIN CLASSIFICATION/STAGING SYSTEM

Stage	**GFR**	**UO**
1	↑ SCr × 1.5-2.0-fold or ≥26.5 µmol/L (≥0.3 mg/dL)	<0.5 mL/kg/h × > 6 h
2	↑ SCr × 2 – 3-fold	<0.5 mL/kg/h × >12 h
3	↑ SCr × >3 fold or if baseline SCr ≥354 µmol/L (≥4 mg/dL) ↑SCr ≥44.2 µmol/L (≥0.5 mg/dL) or on RRT	<0.3 mL/kg/h × >24 h or anuria × >12 h

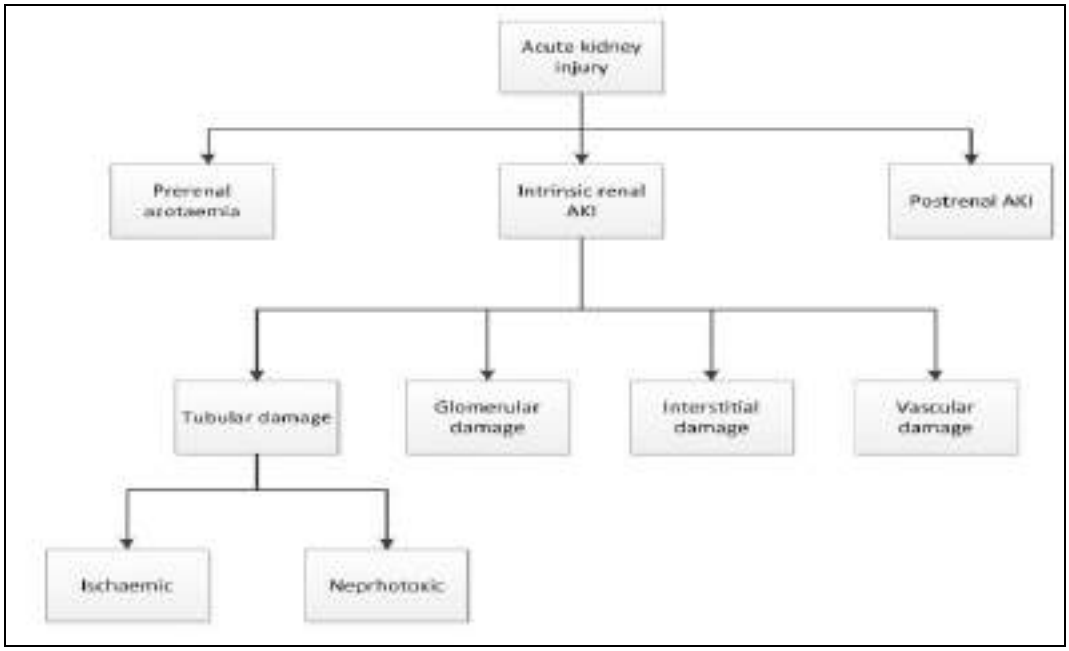
The ability of both the RIFLE and AKIN criteria to predict mortality and severity of patient illness has been validated by numerous studies. However, studies have also shown that reliance on only one of these criteria to the exclusion of the other may miss some cases of acute kidney injury. The Kidney Disease: Improving Global Outcomes (KDIGO) group has therefore combined both the AKIN and ADQI definitions in an attempt to provide a unified consensus definition of acute kidney injury.[11]

The KDIGO definition incorporates the AKIN requirement of an increase in serum creatinine of ≥ 26.5μmol/l within 48 hours. The KDIGO definition also incorporates the ADQI RIFLE criteria in the requirement for an increase in serum creatinine by a multiple of 1.5 from baseline, over 7 days. Additionally, a decrease in urine output by < 0.5ml/kg/hour for > 6 hours also fulfils the requirements for the diagnosis of acute kidney injury. The KDIGO group stages acute kidney injury for severity according to the AKIN criteria.[2]

One additional change in the criteria was made for the sake of clarity and simplicity. For patients reaching Stage 3 by SCr >4.0 mg/dL (>354 μmol/L), rather than requiring an acute increase of ≥0.5 mg/dL (≥44 μmol/L) over an unspecified time period, it instead require that the patient first achieve the creatinine-based change specified in the definition [either ≥0.3 mg/dL (≥26.5 μmol/L) within a 48-h time window or an increase of ≥1.5 times baseline]. This change brings the definition and staging criteria to greater parity and simplifies the criteria.[2]

Acute kidney injury can arise from pre-renal causes such as diarrhoeal illnesses, intrinsic renal causes (such as acute tubular necrosis, acute interstitial nephritis and glomerulonephritis), obstruction, and vascular causes. The detection of the underlying cause of acute kidney injury is important as some causes require prompt specific therapy, such as the relief of urological obstruction, avoiding nephrotoxic agents.

FIGURE 1: CLASSIFYING AKI BASED ON ETIOLOGY



Acute tubular necrosis is the most common cause of acute kidney injury. While the definitive diagnosis of acute tubular necrosis is based on histology, in clinical practice this tends to be according to the clinical setting, urine microscopy (such as the finding of muddy brown granular casts and tubular epithelial cells) and the course.[12,13]

The clinical evaluation of AKI includes a careful history (e.g. Nephrotoxic drug history) and physical examination. Physical examination should include evaluation of fluid status, signs for acute and chronic heart failure, infection, and sepsis. Measurement of cardiac output, preload, preload responsiveness, and intra-abdominal pressure should be considered in the appropriate clinical context.

Laboratory parameters— including SCr, blood urea nitrogen (BUN), and electrolytes, complete blood count and differential—should be obtained. Urine analysis and microscopic examination as well as urinary chemistries may be helpful in determining the underlying cause of AKI. Imaging tests, especially ultrasound, are important components of the evaluation for patients with AKI. Finally, a number of biomarkers of functional change and cellular damage are under evaluation for early diagnosis, risk assessment for, and prognosis of AKI.[2]

Fluid therapy is vital for patients with volume responsive acute kidney injury However, caution should be taken to avoid fluid overload as studies have clearly linked fluid overload to an increased risk of mortality. Fluid resuscitation using synthetic high molecular weight colloids has been linked to acute kidney injury, presumably due to tubular injury from osmotic nephrosis. In the absence of hemorrhagic shock, KDIGO recommends isotonic crystalloids rather than colloids as initial management for expansion of intravascular volume.[14,15]

In critically ill patients, hypotension needs urgent assessment and treatment with IV fluids challenges and vasopressor agents where indicated. Medication should be reviewed with temporary cessation of ACEi and ARBs if already prescribed and nephrotoxic agent like NSAIDs and aminoglycosides should be avoided.[16]

Many patients go on to require renal replacement therapy to treat the complications (such as refractory hyperkalaemia) that cannot be managed by medical therapy alone. The forms of dialysis employed in the treatment of acute kidney injury include intermittent modalities such as intermittent haemodialysis and sustained low efficiency haemodialysis and continuous replacement therapy, such as continuous veno-venous haemodialysis and peritoneal dialysis.[2]

Studies have found no difference in patient outcomes between the use of intermittent haemodialysis and haemodialysis involving continuous renal replacement therapy.[17,18]

The use of peritoneal dialysis for adults is less common in developed countries, but is commonly used in developing countries. It has been demonstrated that this form of cost-effective therapy can be administered in resource limited settings, such as in Moshi, Tanzania, with encouraging results.[19,20]

There is considerable variation worldwide on the indications for and timing of initiation and discontinuation of RRT for AKI. Numerous parameters for metabolic, solute, and fluid control are generally utilized to guide the initiation and discontinuation of RRT. However, there are currently no standards in this field. [21]

One of the major limitations in establishing parameters for optimal timing of dialysis for AKI is that there are no large studies that have demonstrated that timing of dialysis initiation is a factor that determines patient outcomes in AKI. [22]

It is recommended that patients with acute kidney injury receive a calorie intake of 20- 30 kilocalories per kilogram per day, preferably by the enteral route. Negative nitrogen balance in critically ill patients with acute kidney injury is associated with in-hospital mortality. Protein intake of 1.0–1.5 g/kg/d should be kept in patients with AKI on RRT, and up to a maximum of 1.7 g/kg/d in patients on continuous renal replacement therapy to compensate for amino acid loss.[2,23]

Community acquired AKI (CA-AKI) is not a new concept. Some of the earliest case reports of AKI described persons with severe traumatic muscle injuries and subsequent rhabdomyolysis from the battlefield or areas of natural disaster.[24-26] AKI occurring outside of the hospital setting – that is community acquired-AKI (CA-AKI) – has gained increased attention over the last 2-3 years. Hospital acquired-AKI (HA-AKI) has been the focus of research over the last two decades. A definition that maximally utilizes the available clinical information (serum creatinine and urine output) has been developed in this setting of HA-AKI.

In global snapshot of AKI by ISN, majority patients had developed community acquired AKI (60%), and it was about 80% in Low & Low Middle Income Countries (LLMICs), even when rural areas were ill-represented from these countries. It shows the importance of Community acquired-AKI (CA-AKI). [3, 4]

Although all these consensus criteria (RIFLE, AKIN, and KDIGO) have helped standardize the approach to the diagnosis and staging of AKI, there still remain specificity limitations. One of the most discussed limitations is the importance of determining the baseline kidney function, an important aspect for diagnosing and classifying Community acquired AKI. Baseline values are unknown in most of the settings of Community acquired AKI and if they are known they bring their own bias that why it was checked. The lack of a uniform approach to

estimate this baseline has recently been shown to compound the risk for AKI misclassification, hindering effective comparisons of this disease between settings.[27-29] A standard definition for baseline SCr does not exist, leading to heterogeneity across research studies and the potential for misinterpreting the true nature of perturbed kidney function in hospitalized patients.[28,30] The choice of the estimation technique used to obtain the baseline SCr value has an effect on the prevalence of AKI, the severity (or stage) of disease, and on the mortality, risk associated with various stages of AKI.[30]

Several strategies for estimating the basal SCr have been proposed.[29,31,32], ranging from the use of an estimation of SCr by backward calculation from a presumed 'standard GFR' of 75 mL/min/1.73 m2, use of the SCr value at admission, or the peak SCr in the AKI episode under consideration. Whereas RIFLE and KDIGO suggest using the back calculation, AKIN recommends using the evolution of SCr relative to the first observed value in that episode, while the European Renal Best Practice position statement recommend using the admission SCr. However, Siew et al. [29] reported that the use of the admission SCr value as a baseline resulted in a nearly 50% reduction in the detection of AKI compared with that using a known baseline SCr. It appeared that the MDRD-estimated baseline SCr had a significantly higher sensitivity for 'detecting' AKI than the two other methods. Although the use of SCr levels at admission resulted in the least amount of misclassification, this parameter also had the lowest sensitivity (38.9%) for diagnosis of AKI. When admission SCr levels were used, nearly half of the 'true' AKI cases were missed: 25.5% of the cohort had AKI when true baseline SCr levels were used, versus 13.7% with use of admission SCr levels. This decrease is perhaps best explained by the missed diagnosis of community-acquired AKI that improves or stabilizes during hospitalization.[33] Zavada et al. found that use of a Modification of Diet in Renal Disease (MDRD) equation-estimated baseline creatinine could result in either overestimation or underestimation of some mild (Risk) AKI cases but is unlikely to misclassify patients in Injury and Failure RIFLE classes.[32] Thus, it is

important to consider the goal of AKI classification when selecting a baseline. For clinical purposes, it may be important ‘not to miss’ any cases of potential AKI. Therefore, using the MDRD equation to estimate a baseline kidney function, at least provisionally, makes sense. The same may hold true for epidemiologic studies where some degree of overestimation or underestimation may average out and not affect overall rates. On the contrary, for research studies where precise case adjudication is critical, estimated creatinine should be used with caution, if at all, especially for defining mild (stage 1) AKI.[33]

CA-AKI is more common in developing nations including those in tropical zones. This distinction is important because the usual etiologic spectrum of AKI endured in developing countries, especially the tropical setting, is unique, heavily influenced by local climatic and economic conditions, ranging from plant or animal toxin ingestions or infections such as malaria or Hantavirus, to obstetric complications. Previously healthy, relatively young individuals (typically aged 37–47 years) who have developed AKI as a result of factors encountered in their environment dominate the tropical AKI population. [5]

Patients in the tropics continue to confront diseases that are secondary to public-health problems, such as poor sanitation, unsafe drinking water, a lack of infection control and out-of-hospital childbirth. [34]

Water quality has a major role in the ecosystem of disease in the tropics. Tropical soil is fragile; high temperatures and heavy precipitation leach minerals and organic compounds, which enter flowing water, [35] leading to waterlogging and contamination of grain fields. The end result is a high prevalence of water-borne diseases, many of which are associated with AKI. In most patients who develop infection-associated AKI in the tropics, the source of disease is closely linked to contact with contaminated water. The spread of diseases by direct transmission, such as in aerosols is also more likely in the tropics because of overcrowding and poor living conditions.

Tropical ecosystems also favour the proliferation of vermin and parasites. High temperatures, wet weather and high salinity favour the persistence of infection in animal reservoirs, intermediate hosts and

vectors of parasitic diseases, as well as promote the survival of pathogenic micro-organisms outside the human host, thereby increasing the population of vectors and transmission of water-borne disease.[36-38] The tropical climate also affects the incidence of snake bites. Flooding of snake burrows in the rainy (monsoon) season forces their inhabitants to come to the surface at times when large numbers of workers are in the fields for planting or harvesting crops, leading to a spike in the number of bite victims. Seasonal variation in snake envenomation is seen throughout the tropics.[39,40]

Individuals living in poverty are exposed to greater personal and environmental health risks, are less well nourished, have less information and are less able to access healthcare than are affluent individuals. Populations in the tropics are characterized by greater heterogeneity than in temperate climates in terms of socioeconomic status, lifestyle, level of education and access to goods, services and medical care. People living in affluent areas in these countries are less exposed to the environmental risks than those in poor rural areas, but are not completely protected. Moreover, almost all cities in tropical countries contain large slum areas and shanty towns where basic amenities are as limited as those in rural areas, making their disease patterns almost indistinguishable. Poverty and the lack of appropriate regulations also increase the risk of an encounter with industrial toxins that can cause AKI.[41]

The combined effect of a high disease burden and poor economic performance is reflected in the lower life expectancy, higher infant mortality and maternal mortality rates throughout the tropics compared with developed countries. Even after correction for the level of income, the infant mortality rate in the tropical zone is 52% higher and the life expectancy 8% lower than in temperate zones.2 Tropical regions are often characterized by high fertility and high mortality, 2 which result in a high proportion of children in the population, but at the cost of fewer resources per child, limited opportunities for primary and higher education, and a lack of access to primary health care. Conversely, illness reduces productivity and household savings, decreases learning

ability, and leads to a diminished quality of life, thereby perpetuating or even increasing poverty.[42] The developing tropical countries consequently lag behind those of the temperate zone in terms of scientific and technological innovation.[43]

Health-care systems in parts of the tropics are not well developed and specialized care is scarce or distributed unevenly. For cultural reasons, access to health care can be disproportionately restricted in certain sectors of society, for example women and children. Limited access to health care, suspicion of modern medical systems, high health-care costs, and spiritual and cultural beliefs result in continued reliance on traditional medical systems. The use of potentially harmful indigenous tropical remedies contributes to the development or exacerbation of AKI.[44,45] The reliance on non-traditional medical care also contributes to delayed presentation of these individuals to a hospital or treatment centre, which often results in life-threatening complications in those with already established AKI. Economic considerations can also prevent the implementation of technological solutions that might be feasible in more affluent countries; for example, technologically advanced and expensive treatments, such as continuous renal replacement therapy, are eschewed in favour of cheaper and less complex peritoneal dialysis, in developing tropical countries.[46]

Few studies have been published on the epidemiology of AKI in tropical countries.[47] Most published data are from single-centre studies conducted in urban areas and might not reflect the true prevalence of AKI, especially in rural areas;[48] many rural patients who develop AKI do not reach urban hospitals. Furthermore, the available studies have used different denominators to calculate the incidence of AKI, making comparisons difficult.[49-53] Thus, the reported incidence of AKI in tropical countries ranges from 0.31 cases per thousand hospital discharges[53] to 7.9 cases per thousand hospital admissions.[54] Approximately 0.10–0.25% of all admissions to Thai and Indian hospitals are for the management of severe AKI that requires dialysis. In one study, 0.6% of all patients at admission in a US hospital had AKI, but only 5% of them needed renal replacement therapy.[55] AKI is

recognized as the most commonly encountered community-acquired renal emergency in tropical countries.[56,57]

The population of patients with AKI in developing tropical countries is younger (mean age 37–47 years) than that reported in developed temperate countries (mean age 68 years).[55,57-59] Most patients with AKI in developing tropical countries do not have pre-existing comorbidities, such as hypertension, diabetes or chronic kidney disease (CKD), which are characteristically found in patients with AKI in the hospitals of temperate countries but this trend is also changing.

Kaufman and colleagues performed the first formal analysis of CA-AKI.[60] They prospectively identified patients admitted to a single Veteran Affairs (VA) hospital over a 17-month period. The definition of AKI was derived from a schema for hospital acquired acute renal failure used by Hou et al.[61] They classified the majority of CA-AKI as pre-renal. Kaufman et al found that the prevalence of CA-AKI was 1% of hospital admissions, whereas HA-AKI was 4%. This is in contrast to later reports. For example, in the Liano et al study that explored the population level epidemiology of hospital-detected AKI (at or during admission), the major proportion of cases were identified on admission (60%).[62]

In studies that specifically examined the epidemiology of CA-AKI as compared to HA-AKI, CA-AKI has been found to be 2-3x more prevalent than HA-AKI. In these studies, the cases of AKI were identified by international classification diagnostic (ICD) codes for ARF, thereby possibly underestimating the true prevalence of both CA- and HA-AKI in hospital.[63]

Population-wide incidence of CA-AKI was investigated by two separate groups. Liano and colleagues noted that of the total AKI identified in the hospital setting, 60.4% were identified on admission; however, this study did not distinguish the incidence of CA-AKI from HA-AKI in their final analysis.[62]

Talabani and colleagues conducted the first population-wide analysis of changes in serum creatinine values specifically examining all variety of CA-AKI.[64] Of the patients who had creatinine measurements in an

outpatient laboratory during a one month period, 0.7% were identified as meeting the criteria for CA-AKI (50% rise from baseline serum creatinine – which was measured in the 6 months prior to the index creatinine value. As compared to a random subset of the population who did not suffer CA-AKI, the CA-AKI individuals were older (70 v 57 yrs.) and more likely to have pre-existing CKD (37.8% v 27.7%, p=0.023).

Der Mesropian et al examined renal outcomes of patients with either CA- or HA-AKI as compared to patients who had no AKI.[65] Twelve percent and 6.1% of those with CA-AKI developed a doubling of serum creatinine or end stage renal disease at 3 years, respectively. These estimates were similar in patients with HA-AKI. Mortality was also very common among patients with any AKI and not different between the two groups. This was different from the findings of Wonnacott et al who looked at mortality at 14 months following CA-AKI. As compared to HA-AKI, patients with CA-AKI demonstrated a higher survival rate (55% v 36.9%, p<0.001).[66]

In another study by Yafang Wang et al, a nationwide study on community acquired AKI in China, 4,136 patients were identified during the 2-month snapshots (January 2013 and July 2013). Of these, 2,020 (48.8%) had cases related to decreased kidney perfusion; 1,111 (26.9%), to intrinsic kidney disease; and 499 (12.1%), to urinary tract obstruction. In the north versus the south, more patients were exposed to nephrotoxins or had urinary tract obstructions. 536 (13.0%) patients with community-acquired AKI had indications for renal replacement therapy (RRT), but only 347 (64.7%) of them received RRT. Rates of timely diagnosis and appropriate use of RRT were higher in regions with higher per capita gross domestic product. All-cause in-hospital mortality was 7.3% (295 of 4,068). Delayed AKI recognition and being located in northern China were independent risk factors for in hospital mortality, and referral to nephrology providers was an independent protective factor. They concluded that features of community-acquired AKI varied substantially in different regions of China and were closely linked to the environment, economy, and medical resources.[67]

In 2004 Biradar V et. al studied the clinical profile, etiology and outcome of patients developing renal failure in the hospital and also compared the outcome in patients with HAARF with that of patients with community acquired acute renal failure (CAARF). One hundred and thirty-seven (137) consecutive patients of HAARF were evaluated and compared with five hundred and five patients with CAARF as controls. Patients with HAARF were significantly older compared to patients with CAARF. Drugs were the commonest factor responsible for HAARF (38%) followed by sepsis (35%). Patients with HAARF were sicker than compared to patients with CAARF. Mortality rate was 51% compared to 20%. Their study highlighted the higher morbidity and mortality among HAARF patients compared to CAARF patients.[68]

In 2013 Schissler MM et.al compared the prevalence, causes, severity and outcomes of patients with CAAKI and HAAKI. AKI was verified by applying the RIFLE criteria. Four hundred twenty- two patients met inclusion criteria, of which 335 (79.4%) developed CAAKI. Patients with CA- AKI were more likely to have volume depletion as the etiology, had fewer chronic illnesses and hospital complications, had a shorter length of stay, and had a reduced mortality, compared with HAAKI. Distribution among the three RIFLE classes did not differ between groups, and recovery of renal function was incomplete in both groups. They concluded that CAAKI is a common cause of AKI that is as severe as that seen in HAAKI. CAAKI has a significant impact on length of hospital stay, mortality, and the development and/or progression of chronic kidney disease.[55]

In 2014 Eswarappa M et.al described the epidemiology, etiology, clinical characteristics and outcome of AKI in 500 critically ill patients without pre-existing renal disease, diagnosed using RIFLE criteria. One fourth of patients developed HAAKI and remaining presented with CAAKI. AKI was predominantly encountered in older males. Diabetes, hypertension, coronary artery disease were the most commonly prevalent comorbidities. Sepsis was the most common cause of AKI, accounting for 38.6% of patients. 24.4% belonged to risk class, 37.0% to injury class, 35.0% to failure class, 3% to loss and 0.6% to ESRD

class of the RIFLE criteria. Renal replacement therapy was required in 37.2% of patients. About 60% recovered complete renal function. CKD was a sequel in 2.4% of patients. Average duration of ICU stay was 5.6 days. Crude mortality rate was 37.6%. In critically ill patients without pre-existing renal disease, elderly age, male sex, type 2 diabetes along with a primary diagnosis of sepsis were most commonly associated with AKI.[69]

In 2015 Bardai G et.al compared epidemiology, clinical characteristics, etiologies, severity and outcomes of patients of these HAAKI and CAAKI. Among the 210 patients with AKI, 157 were classified as CAAKI (74.8%). There was no significant difference in age average and comorbidities between CAAKI and HAAKI. Dehydration and volume depletion were significantly more prevalent in patients with CA-AKI (47.7% vs 34%). HAAKI was associated with a significantly higher prevalence of acute tubular necrosis than CAAKI (50% vs. 3.8%). There were no significant differences between the numbers of patients requiring renal replacement therapy, and the length of hospital stay in both groups. The mortality in hospital was significantly higher in the HAAKI group compared to CAAKI group (39.6% vs. 25.4%).[70]

In a study done by S. Goswami et al in an urban center of North India, CA-AKI was more common than HA-AKI (57.2% vs 42.8).[71] There was no significant difference in age average and co morbidities between CAAKI and HAAKI. Dehydration and volume depletion were significantly more prevalent in patients with CAAKI (47.9% vs. 28.97% for HAAKI $p<0.0001$). While HAAKI was associated with a significantly higher prevalence of drug induced AKI (23.36% vs. 7% in CAAKI $p<0.0001$). The mortality in hospital was significantly higher in the HAAKI group compared to CAAKI group (51% versus 20% $p<0.0001$). This study highlights that risk factors for CAAKI and HAAKI are similar, with pre-existing CKD, diabetes, heart disease, hypertension, cancer and tuberculosis being common risk factors for both.

In a retrospective study done by Anupma Kaul et al in an urban centre of North India, where they excluded CKD and Diabetic patients, 2.5% patients with a mean age of 39.8 were diagnosed to have CA-AKI.[72]

The most common cause of CA-AKI was medical (77.5%), followed by obstetrical (14.2%) and surgical (8.3%) causes. Among the medical causes, acute diarrheal disease was the most common cause (29%), followed by malaria (18.8%) and sepsis (13.9%). Sepsis had the highest in-hospital mortality (46%). Nephrolithiasis was the most common surgical cause. Puerperal sepsis (44.1%), pre-eclampsia (23.5%), intrauterine death (11.8%), antenatal hemorrhage (11.8%) and post-partal hemorrhage (8.8%) were the obstetric causes.

In 2016 Hsu CN et al retrospectively investigated the incidence, outcomes, and risk factors of AKI in a large Taiwanese adult cohort. 734340 Patients with diagnosis of AKI at discharge were classified as either CA- or HA-AKI based on the RIFLE classification criteria. The incidence of CA-AKI was 17.25 and HA-AKI was 8.14 per 1000 admissions. Patients with HAAKI had higher levels of in hospital mortality as compared to HAAKI (51.58% vs. 26.07%). Risk profile was different between two groups with preexisting chronic diseases being more common in CAAKI as compared to HAAKI. Patients with HA-AKI had more severe outcomes than patients with CA-AKI, and demonstrated different spectrum of risk factors like male sex, cancer history, nephrotoxic medicines.[73]

In 2017 Wang S et.al compared the long-term outcomes of hospital and community acquired acute kidney injury. The study cohort consisted of 352 patients (139 for HAAKI and 213 for CAAKI). The 1-year all-cause mortality was significantly different between patients with HAAKI and CAAKI (56.8% vs. 41.8%), but the 3-year all-cause mortality showed no significant differences between the two groups (64% of HAAKI and 60.1% of CAAKI). They concluded that there was no difference in 3-year all-cause mortality between HA-AKI group and CA-AKI group.[74]

In 2017 Patel M et.al assessed for the clinico-etiological aspects and outcome of AKI in north tribal Maharashtra. 110 patients with clinical and laboratory evidence of renal dysfunction were included. Patients who developed kidney injury after at least 24 hours of admission were referred as hospital acquired HAAKI. Patients who had AKI on

admission were considered as community acquired CAAKI. Out of 110 patients, 85.45% had CAAKI and 14.54% had HAAKI. Overall mortality was 39% with higher in CAAKI as compared to HAAKI (34.5% VS. 4.5%). Comparing survivors with non-survivors, it was found that mortality was associated with delayed presentation, presence of hypotension, creatinine, and need for vasopressors and higher Sequential Organ Failure Assessment (SOFA) score. They concluded that in this tribal area CAAKI had higher mortality than HAAKI. The most common etiologies were complicated malaria and obstructive pyelonephritis in this tribal part of the country. Their study highlighted that presence of hypotension, SOFA score >11, oliguria and multi-organ dysfunction are the risk factors for mortality.[75]

In 2017 Aggarwal HK et.al assessed the spectrum and outcome of acute kidney injury. A total of 2777 patients in the medicine, surgery, and obstetrical wards were screened for AKI as per KDIGO guidelines from 1 April 2015 to 31 December 2016. Of the total 112 AKI patients, 94 had CAAKI (83.92%) while 18 (16.7%) had HAAKI. 60 patients were hemodialysed (12 HAAKI and 48 CAAKI). Mortality in HAAKI was 16.6% while in CAAKI it was 22.34%. The majority of patients (53.33%) were from the medical specialty, in which sepsis and gastroenteritis with hypovolemia were the main causes. Regarding the final outcome for the AKI patients, 59.82% patients recovered with normal renal function, 21.42% patients died, and 18.75 patients developed CKD. From the results, they concluded that AKI is associated with high mortality and morbidity if not managed promptly.[76]

In 2018 Hsu CN et.al assessed epidemiology and outcomes of CAAKI and HAAKI in children and adolescents. Mortality was higher among AKI patients than in no-AKI patients with higher in HAAKI as compared to CAAKI (13.64% vs. 3.7%). Hospital length of stay was longer for HAAKI (median 24 days) as compared to CAAKI (median 7 days) and increased with increase in severity of AKI. Highest mortality (9%) was in age group of 13 to < 18 years (HAAKI: CAAKI = 6%: 3%). Patients with AKI and those with severe stages of AKI resulted in an increase in health-care service utilization. They concluded that increasing

incidence of AKI in the emphasizes the need for an increased awareness of AKI among health professionals to identify at-risk children and monitor SCr, so that modifiable risk factors can be managed.[77]

Kohli H S et al in 2007 did a prospective study on Predictors of Mortality in Acute Renal Failure in a Developing Country. Of 33,301 patients admitted during the study period, 294 (0.88%) were either admitted with or developed ARF after hospitalization. Mean age was 43.9 ± 16.9 (18–86 yrs). Sepsis was the most common cause (63.26%). Pre-existing diseases like cardiovascular disease (CVSD), respiratory system disease (RSD), central nervous system disease (CNSD), hypertension, diabetes mellitus (DM), and malignancy were significantly higher in elderly as compared to younger patients. On univariate analysis sepsis, hypoperfusion as a cause of ARF and hospital-acquired ARF were associated with higher mortality. Pre-existing diseases viz. RSD, CVSD, CNSD, and DM had higher mortality. Among the severity and complications of ARF, oliguria, bleeding and infection during the course of ARF and critical illness were predictors of poor outcome. Age >60 yrs was associated with significantly

higher mortality. However, on multivariate analysis, only critical illness (odds ratio 37.3), age > 60 years (odds ratio of 5.6), and sepsis as cause of ARF (odds ratio of 2.6) were found to be independent predictors of mortality.[78]

In 2012, Kumar S et al published Spectrum of acute kidney injury in the Himalayan region. Over a period of 1 year, 102 patients with clinical and laboratory evidence of azotemia were included. Out of 102 patients, 84.3% had community acquired AKI and 15.7% had hospital acquired AKI. Medical causes were leading contributors (85.3%), with septicemia being the main factor (33.3%). Multiorgan failure was present in 59.8% patients. The overall mortality was 29.2%, and community acquired AKI was associated with higher mortality as compared to hospital acquired AKI (22.5% vs 6.7%). study highlighted Septicemia, volume depletion and nephrotoxins as the leading cause of

AKI and association of hypotension, multiorgan failure and oliguria with mortality.[79]

In 2017, Inokuchi R et al did a systematic review and meta-analysis on Differences in characteristics and outcomes between community- and hospital-acquired acute kidney injury. Eight eligible observational studies with 17,939 patients were included. The systematic review showed that patients with CA-AKI had a significantly higher rate of AKI stage 3 compared with those with HA-AKI. The meta-analysis revealed that patients with CA-AKI showed significantly lower in-hospital mortality (odds ratio (OR) = 2.79; Z = 8.22; 95% confidence interval (CI), 2.18 – 3.56; $p < 0.001$) and shorter length of hospital stay (OR = 2.55; Z = 5.81; 95% CI, 1.86 – 3.50; $p < 0.001$). Subanalyses of studies conducted in developing and developed countries showed that patients with CA-AKI had lower in-hospital mortality and shorter length of hospital stay. Jackknife sensitivity analyses confirmed the replicability of these findings ($p < 0.001$). The results indicate that significant differences exist in clinical phenotypes between CA- and HA-AKI, even when standardized diagnosis criteria are used.[80]

IN 2019, Kaaviya et al did a Prospective observational study on CA-AKI in Southern India to study the etiology, clinical characteristics, and short-term outcome of CAAKI in adults using 2012 KDIGO AKI criteria. The study did not include obstetric or surgical cases of AKI. Of 186 CAAKI patients (mean age, 46.13 ± 15.2 years), 86/186 was infective etiology, 93/186 was noninfective etiology, 7/186 was due to intrinsic renal pathology. Pyelonephritis 33/186 (17.7%) was the most common infective etiology, and snakebite in 49 (26.3%) was the most common non-infective etiology; 28/186 (15.1%) died. On logistic regression, hypotension, mechanical ventilation, thrombocytopenia, and anuria were associated with mortality.[81]

In 2019, Vikrant S et al, did a study on Epidemiology and Outcome of Acute Kidney Injury. Three hundred and nine patients had AKI. The observed incidence of AKI was eight per 1000 admissions. About 92.2% had community-acquired AKI (CA-AKI), and in 7.8% it was hospital-acquired AKI (HA-AKI). Etiological factors for AKI were

medical in 87.4% of the cases, surgical in 9.4%, and obstetric 3.2%. Sepsis was the most common (53.1%) etiology of AKI among the medical cases. Among sepsis, scrub typhus, urosepsis, and pneumonia were the most common causes of AKI. Hypovolemia (9.4%), biological toxins (8.4%), nephrotoxic drugs and chemicals (7.4%), cardiac causes (7.4%), and acute glomerulonephritis (1.9%) were other medical causes of AKI. Nearly 38.2% had multiorgan failure, 20.1% required vasopressors, 6.1 % required Intensive Care Unit support, and 23.3% required dialysis. Mortality was 8.7%. Anemia, use of vasopressor drugs, and need for intensive care support were independent predictive factors for mortality. AKI is largely CA-AKI and the lesser percentage is due to HA-AKI. Many causes are potentially preventable. Early fluid resuscitation, effective antiinfective treatment, appropriate antidotes, and timely referral of established AKI patients to centers with dialysis facilities can improve AKI outcomes.[82]

It is important to understand that epidemiology of CAAKI will be influenced by the changes in the epidemiology of infections causing it. For example, malarial AKI, which was seen predominantly in *Plasmodium falciparum* infections, is being increasingly seen in *Plasmodium vivax* infections.[83,84] Sethi et al have demonstrated an increasing incidence of leptospirosis in northern India from 11.5% in 2004 to 20.5% in 2008. Sixty percent of the 86 cases included in their study had AKI.[85]

A significant trend changes in the epidemiology of CAAKI are being observed in the past years.

Chugh K.S. et al in 1989 reported the Changing Trends in Acute Renal Failure in Third-world Countries. Of the 1862 patients with acute renal failure requiring dialysis between 1965 and 1986 in a north Indian centre, 60,15 and 25 per cent were related to medical, obstetrical and surgical conditions respectively. Among the medical patients, diarrhoeal diseases which caused 23 per cent of the total number of cases of acute renal failure in the period 1965 to 1974 caused only 10 per cent in 1981 to 1986. In the same period, acute renal failure due to sepsis and drugs increased while that due to copper sulphate

poisoning and intravascular haemolysis showed a downward trend. Obstetrical acute renal failure declined from 22 per cent in 1965 to 1974 to 9 per cent during the period 1981 to 1986. This decline was chiefly due to a fall in cases of septic abortion, puerperal sepsis and postpartum haemorrhage. Surgical acute renal failure increased from 11 per cent during the period 1965 to 1974 to 31 per cent in the 1980s, predominantly due to an increase in patients with obstructive uropathy. They concluded that despite these favourable trends, the pattern of acute renal failure in the third world continues to be different from that in the developed countries.[56]

Prakash J et al, reported the Changing epidemiology of community-acquired acute kidney injury from eastern india. This study aimed at describing changes in epidemiology of community-acquired AKI (CAAKI) over a time span of 26 years in two study periods, namely, 1983–95 and 1996–2008. they studied 2405 (1375 male and 1030 female) cases of AKI in the age range 1–95 (mean: 40.32) years. The incidence of CAAKI in 1983–95 and 1996–2008 was 1.95 and 4.14 per 1000 admission, respectively ($P < 0.01$). Obstetrical AKI has decreased because of the declining number of post-abortal AKI. Surgical AKI decreased from 13.8% in 1983–95 to 9.17% in 1996– 2008($P < 0.01$). Malarial AKI increased significantly from 4.7% in the first half of the study to 17% in the later period ($P < 0.01$). Diarrhea-associated AKI had significantly decreased from 36.83% in 1983–95 to 19% in 1996–2008 ($P < 0.01$). Sepsis-related AKI had increased from 1.57% in 1983– 95 to 11.43% in 1996–2008 ($P < 0.01$). Nephrotoxic AKI showed an increasing trend in recent years ($P < 0.01$) and mainly caused by rifampicin and NSAIDs. Liver disease-related AKI increased from 1.73% in 1983–95 to 3.17% in 1996–2008 ($P < 0.01$). Myeloma-associated acute renal failure (ARF) accounted for 1.25% of the total number of ARF cases in the period 1996–2008. HIV infection contributed to 1.65% of ARF of the total number of AKI cases in the second period (1996– 2008). Incidence of renal cortical necrosis (RCN) decreased significantly from 5.8% in 1983–95 to 1.3% in 1996–2008 of the total number of ARF cases ($P < 0.01$). However, during the same

period ARF due to acute tubular necrosis, acute glomerulonephritis and acute interstitial nephritis remained unchanged. The mortality rate from AKI decreased significantly from 20% in 1983–95 to 10.98% in 1996–2008 ($P < 0.01$). They concluded that epidemiological characteristics of CAAKI have changed over the past three decades. There has been an increase in the overall incidence of ARF with the changing etiology of AKI in recent years. Incidences of obstetrical, surgical and diarrheal AKI have decreased significantly, whereas those of AKI associated with malaria, sepsis, nephrotoxic drugs and liver disease have increased. RCN has decreased significantly. In contrast to developed nations, community acquired AKI is more common in developing countries. It often affects younger individuals and is caused by single and preventable diseases in developing countries.[86]

In 2006, Jayakumar M et al reported epidemiologic Trend Changes in Acute Renal Failure from South India. A total of 1112 patients were diagnosed to have ARF from 1995–2004. The mean age was 37.08 ± 3.4 yrs. There were 669 (60.1%) males. Medical, obstetric, and surgical causes accounted for 87.6, 8.9, and 3.4 percent of ARF, respectively. Among the medical causes of ARF, acute diarrheal disease was the most common. Other causes of medical ARF included drugs, glomerulonephritis, sepsis, snake bite, leptospirosis, malaria, and copper sulphate, which accounted for 13.4, 9.3, 8.8, 7.8, 7.5, 4.4, and 4.3 percent, respectively. In comparison with the data from 1987–1991, medical ARF remained the most common cause of ARF, though without any statistical significance (87.6 percent vs 89.5 percent, $p>0.32$). Though surgical ARF had more than doubled from 1.5 percent from 1987–1991 to 3.4 percent ($p<0.01$) during the present study, it is much less when compared to similar studies in the literature. Obstetric renal failure more or less remained the same (8.9 percent vs 9 percent, $p>0.4$). A statistically significant decline was noted in overall as well as individual group mortality. The overall mortality declined from 26.4 percent to 19.6 percent ($p<0.02$). Regarding the outcome of ARF, 611 patients (54.94 percent) showed a total recovery, a partial recovery was noted in 192 patients (17.26 percent), and 91 patients (8.18

percent) had persistent dialysis-dependent renal failure. The factors noted to occur more frequently in the deceased were high entry serum creatinine (>440 μmol), jaundice, sepsis, oliguria, anemia, hypoalbuminemia, and hospital-acquired ARF. The overall requirement of dialysis was 69.0 percent. Hemodialysis was the most common modality of renal replacement therapy. They concluded that there are significant trend changes with time. Acute diarrheal disease was the most common cause of ARF. Leptospiral ARF was on the decline, and drugs, sepsis, and malaria were the emerging ARF causes. The incidence of surgical ARF was on the rise. Despite improvements in antenatal care, obstetric renal failure remained a significant cause of ARF. Hemodialysis became the preferred mode of renal replacement therapy.[57]

Despite our sophisticated understanding of the pathobiology of acute kidney injury, current prevention strategies are inadequate and current treatment options outside of renal replacement therapy are nonexistent.

International Society of nephrology has recognized acute kidney injury as a major health concern and it aims to curb down mortality associated with acute kidney injury to zero by 2025 (0 by 25 initiative) Knowledge of incidence and risk factors is crucial because it drives local and international efforts on detection and treatment. Also, in developing countries, incidence varies seasonally; incidence peaks cause critical shortages in medical and nursing personnel.

Limited data are available on the etiology and outcome of Community acute kidney injury and their trends in this geographic area. In this observational study, we evaluated the clinical characteristics, etiologies, and outcomes of patients admitted to the hospital with community acquired acute kidney injury.

MATERIAL AND METHODS

This is a prospective analysis of all patients who are admitted to the Dayanand Medical College and Hospital, Ludhiana, India from March 2018 to June 2019. It is a observational study and included all patients during the study period who fulfill inclusion criteria.

SOURCE OF DATA: Indoor patients of Dayanand Medical College.

METHODS OF COLLECTION OF DATA:

INCLUSION CRITERIA

- Patients $\geq$ 18 years admitted to Dayanand Medical College with Acute Kidney Injury at admission or within 48 hours of admission.

EXCLUSION CRITERIA

- Age <18 years and >70 years
- Referred following a hospital-acquired renal insufficiency
- Consent refusal
- CKD Stage 5 D and Post-renal Transplant patients
- Particulars, detailed history and thorough clinical examination was noted in a pre-structured proforma after written consent.

Defining CA-AKI

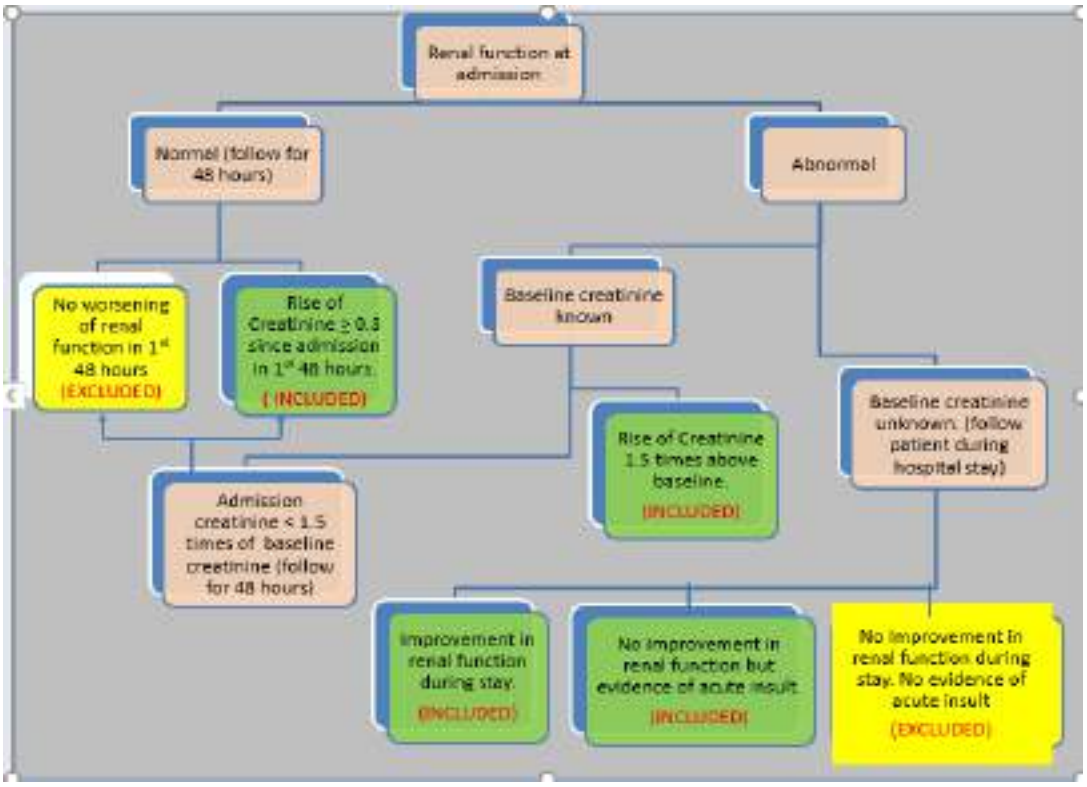
AKI was defined as per KDIGO guidelines.

If the SCr value at admission increases by 1.5 times from the baseline, It was labelled as Community acquired acute kidney injury (CA-AKI). If SCr value increases by $\geq$ 0.3 mg/dl over 1st 48 hours of admission, it also was labelled as CA-AKI.

Baseline SCr was defined as the most recent measurement of SCr within the 3 months prior to admission.

For patient without baseline creat., an increased SCr level at admission and a trend of decreasing SCr levels during the hospital stay or evidence of acute insult in cases of non-resolution was labelled as CA-AKI.

FIGURE 2: STUDY PLAN


Renal function at admission
Normal (follow for 48 hours)
Abnormal
No worsening of renal function in 1st 48 hours (EXCLUDED)
Rise of Creatinine ≥ 0.3 since admission in 1st 48 hours (INCLUDED)
Baseline creatinine known
Baseline creatinine unknown. (follow patient during hospital stay)
Rise of Creatinine 1.5 times above baseline (INCLUDED)
Admission creatinine < 1.5 times of baseline creatinine (follow for 48 hours)
Improvement in renal function during stay. (INCLUDED)
No improvement in renal function but evidence of acute insult (INCLUDED)
No improvement in renal function during stay. No evidence of acute insult (EXCLUDED)

OUTCOME:

Recovery of kidney function at discharge/follow up was classified as

Full recovery- Serum creatinine level decreased to the threshold serum creatinine level, which was defined as ~ 10% of the baseline serum creatinine level.

Partial recovery- Serum creatinine level decreased by > 25% from the peak level, but remained above the threshold.

Failure to recover- the patient is dialysis dependent or scr level decreased by < 25% from the peak level.

Treatment withdrawal refers to patients with AKI who were recorded as severely ill but were discharged because they gave up further treatment in the hospital due to economic or personal reasons.

STATISTICAL ANALYSIS

Continuous data was presented as mean and standard deviation (mean ±Sd) and categorical data as a percent and 95% Confident Interval (CI). At the univariate analysis, proportions were compared using chi-squared test or Fisher exact test and in multivariate analysis, using multiple logistic regression. Continuous data was compared

using t-test and multiples means were compared using AVOVA. Kaplan-Meier survival analysis was used for patient survival. All the statistical analysis was done using statistical software SPSS 21. “p-value” <0.05 was considered as significant.

OBSERVATIONS AND RESULTS

This observational study for epidemiology of community acquired AKI was carried out from March 2018 to July 2019.

During study period a total of 54285 adult patients were screened out of which 790 (1.6%) were suspected to have AKI at admission or within 48 hours of admission (inclusion criteria). Of these 790 patients, 264 were referred from other health facility after developing hospital acquired AKI and were excluded. 63 patients were above age 70, 25 patients were CKD-5D, 10 were post-renal transplant and were excluded. A total 0f 380 cases were finally included in the study for analysis. Community acquired AKI was seen in 7 patients per 1000 admission during the study period Patient flow diagram is in Figure.

TABLE 3: AGE DISTRIBUTION OF THE PATIENTS

Age group	No. of cases	%
<30 years	47	12.4%
30-39 years	53	13.9%
40-49 years	127	33.4%
50-59 years	105	27.6%
>60 years	48	12.6%

Mean age of the study group was 45.4 ± 23.6 years with a median of 47 years.

FIGURE 3: AGE DISTRIBUTION OF THE PATIENTS

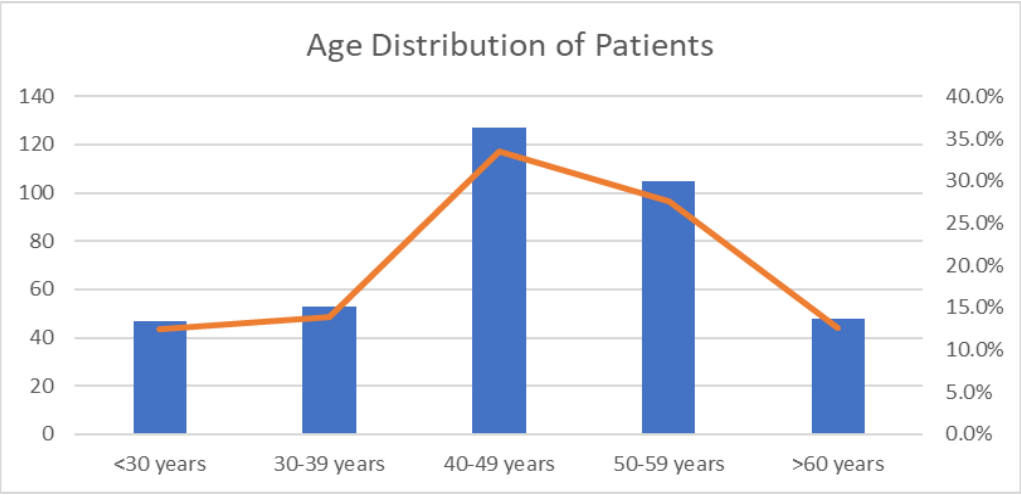


FIGURE 4: STUDY FLOW DIAGRAM

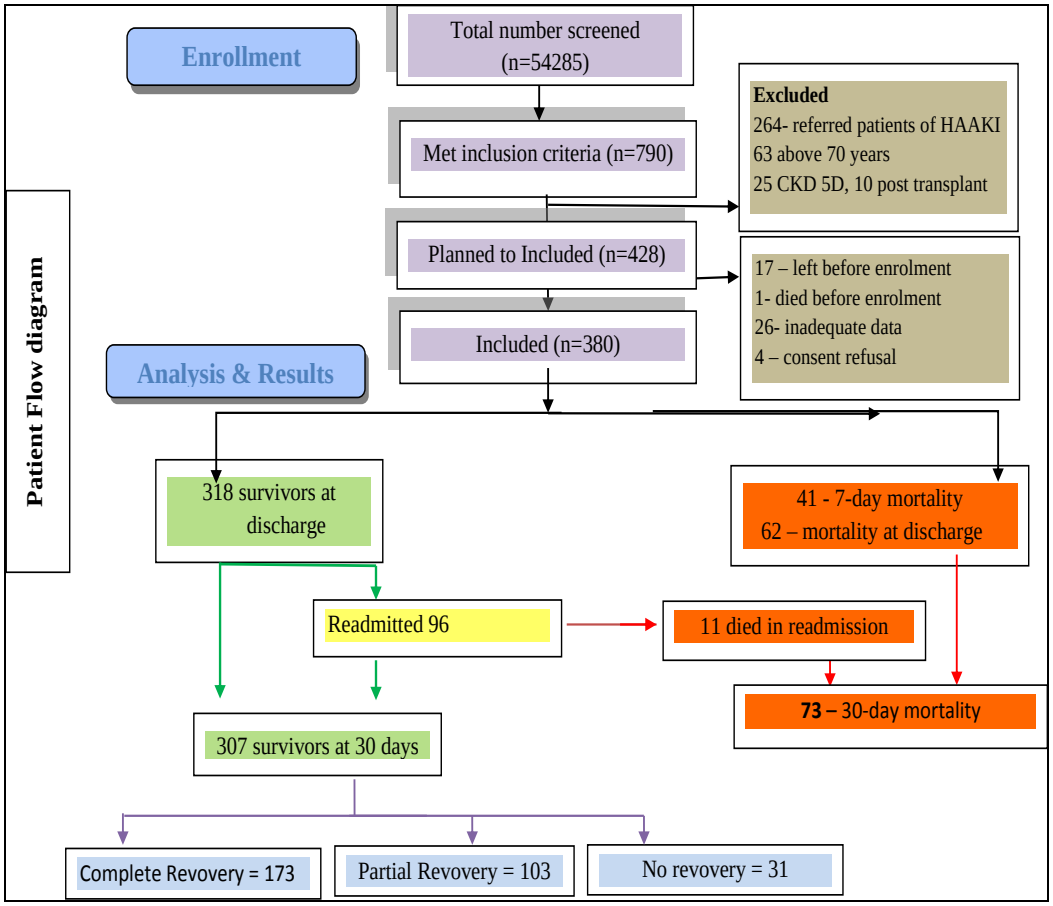


TABLE 4: GENDER WISE DISTRIBUTION OF THE PATIENTS

Gender	No. of cases	%
F	158	41.6%
M	222	58.4%
Total	380	100.0%

41.6% of the cases were female with mean age of 43.8 years while 58.4% cases were male with mean age of 46.6 years. Male to female ratio was 1.4 :1.

FIGURE 5: GENDER WISE DISTRIBUTION OF THE PATIENTS

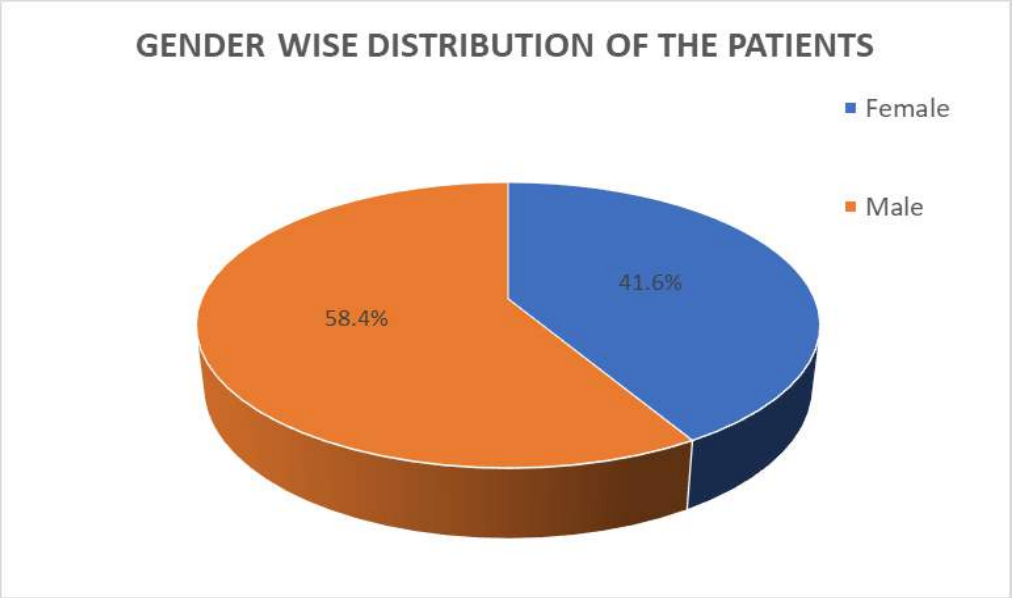


TABLE 5: DISTRIBUTION ACCORDING TO RESIDENTIAL STATUS

Rural/Urban	No. of cases	%
RURAL	221	58.2%
URBAN	159	41.8%

58.2 % people came from a rural area with mean age of 44.8 years while 159 patients were urban dwellers with mean age of 46.3 years

FIGURE 6: DISTRIBUTION ACCORDING TO RESIDENTIAL STATUS

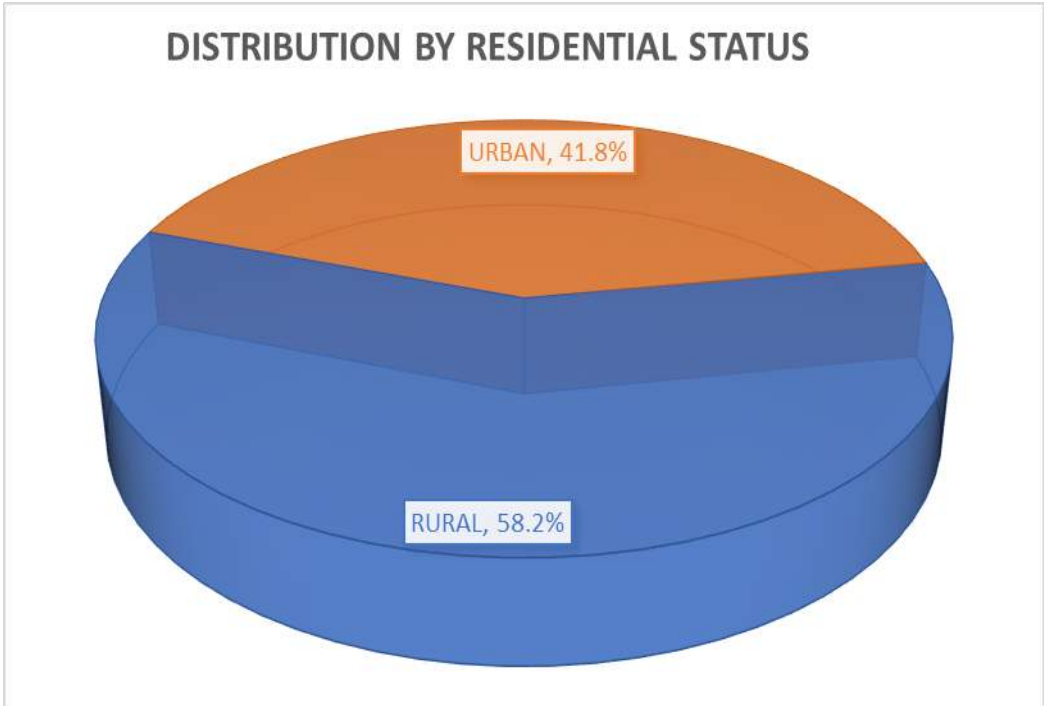


TABLE 6: VARIOUS CO-MORBIDITIES IN THE STUDY GROUP

Comorbidity	No. of cases	%
None	163	42.6%
DM	61	16.0%
HTN	119	31.3%
DM + HTN	25	6.5%
CKD	106	27.9%
CAD	28	7.4%
CVA	19	5.0%
COAD	22	5.8%
CLD	30	7.9%
MALIGNANCY	9	2.4%
TB	14	3.7%
RA	5	1.3%
HBV	2	0.5%
HCV	7	1.8%
HIV	2	0.5%
NEPHROLITHIASIS	14	3.7%

Hypertension was present in 31% of cases while diabetes was present in 16% (61) cases. 26 diabetic patients had evidence of microvascular complications. 13% patient had known chronic kidney disease while 15% more patients were diagnosed to have CKD after evaluation during study period. 6.57% patient had both diabetes and hypertension. 42% patients didn't have any comorbidities. 7.4% patients were known to have coronary artery disease while 5% had history of stroke. 5.8% had chronic obstructive airway disease and 7.9% had underlying chronic liver disease. 2.4% patients had history of treated and active malignancy. 3.7% had active or treated tuberculosis and 3.7% had nephrolithiasis. 5 patients had rheumatoid arthritis, 7 had positive serology for HCV, 2 for HBV and HIV each. Patients with DM, HTN, RA. CAD, CVA, CKD, COAD, CLD and Malignancy had their mean age above 50 with maximum for malignancy (59 years) and CAD (58 years).

FIGURE 7: VARIOUS CO-MORBIDITIES IN THE STUDY GROUP

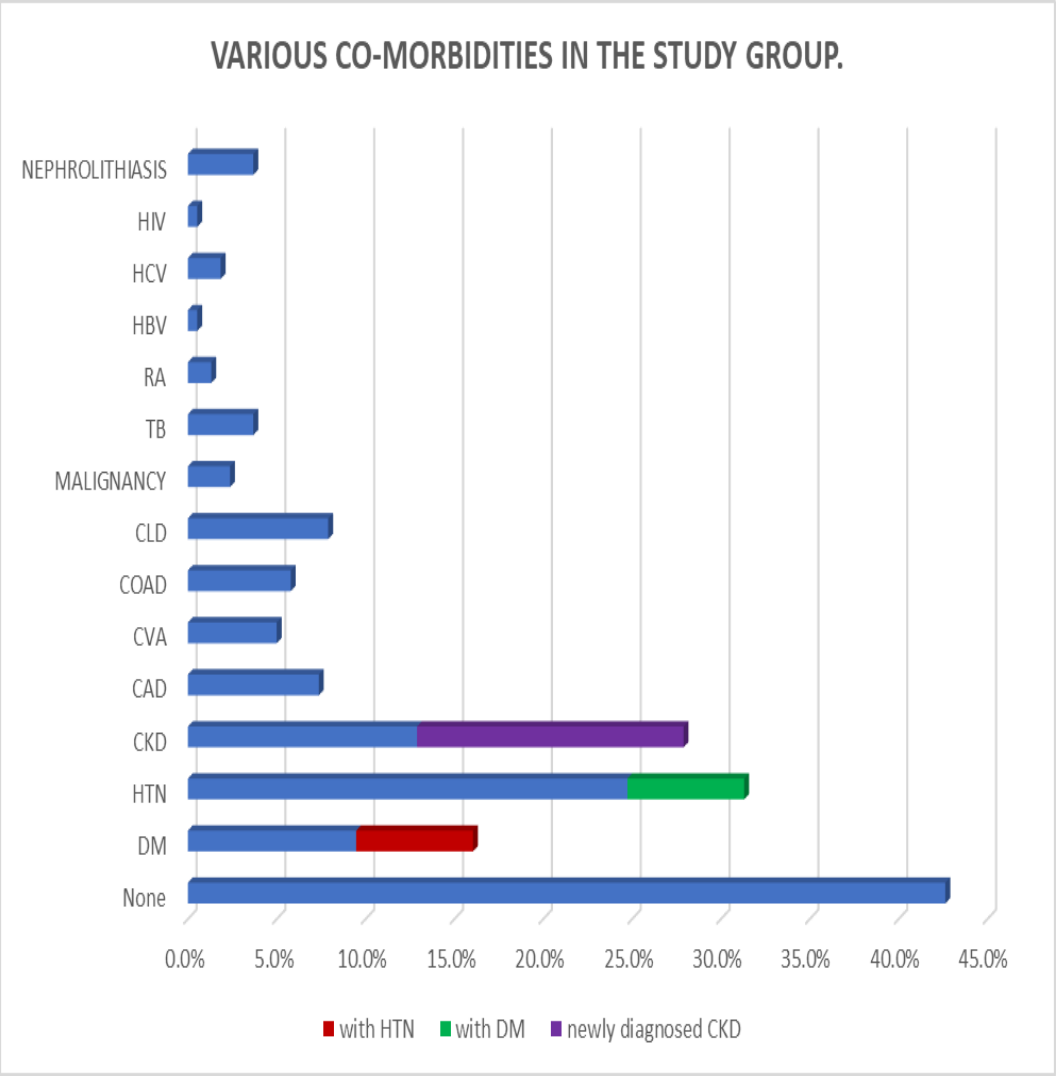


TABLE 7: NUMBER OF COMORBIDITIES AS PER AGE

Age group	NONE	SINGLE	TWO	THREE OR MORE	p-value
<30 years	44 (27%)	0 (0%)	1 (1.3%)	1 (2.8%)	0.001
30-39 years	44 (27%)	6 (5.6%)	1 (1.3%)	2 (5.7%)	
40-49 years	54 (33%)	42 (39.2%)	24 (32%)	7 (20%)	
50-59 years	16 (9.8%)	48 (45%)	31 (41.3%)	10 (28.5%)	
>60 years	4 (2.4%)	11 (10.3%)	18 (24%)	15 (42.8%)	

With increasing age, the number of co-morbidities increased and this was statistically significant with p value below 0.01. Majority of the people with age above 50 years who developed AKI had 1 or more comorbidity.

FIGURE 8: NUMBER OF CO-MORBIDITIES AS PER AGE

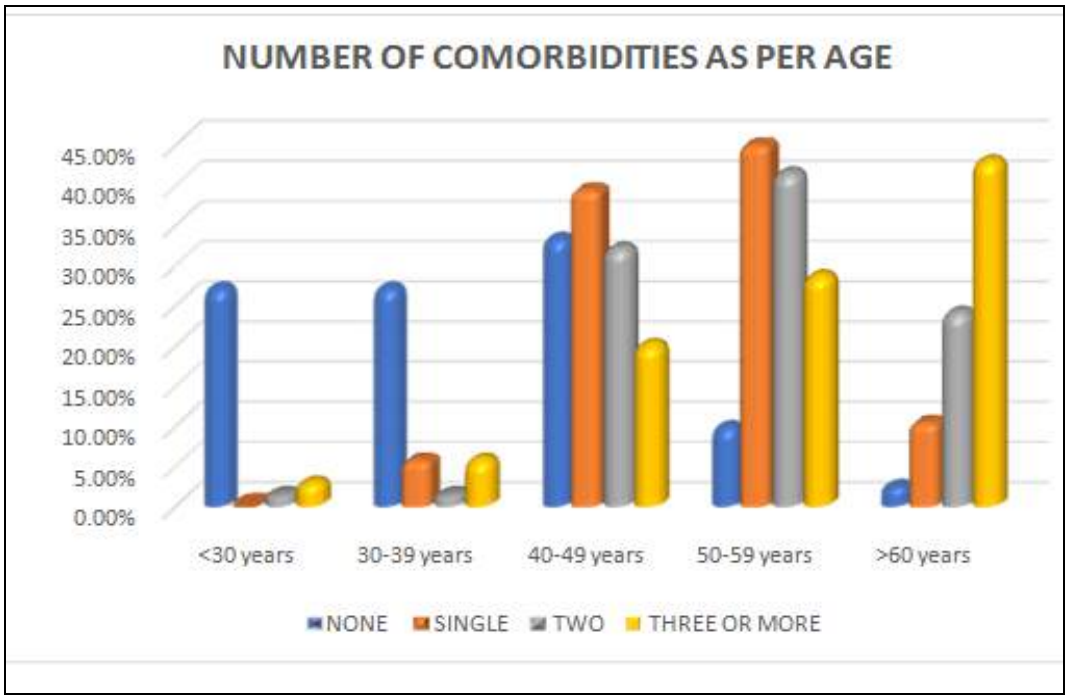


TABLE 8: MEAN AGE FOR COMORBIDITY GROUPS

COMORBIDITY GROUP	mean age ± 2 SD (years)
None	37.3 ± 21.28
Single	51 ± 14.16
Two	51.7 ±16.26
≥Three	55.7 ±21.9

There is contrasting difference between mean age of patients with and without comorbidities. Mean age without comorbidities was 37 years while mean age with even one comorbidity was above 50 years. This was statistically significant using one-way AVONA for group with no comorbidity vs all the other groups separately (p-value below 0.01). There was no difference between other three groups.

FIGURE 9: MEAN AGE FOR COMORBIDITY GROUPS

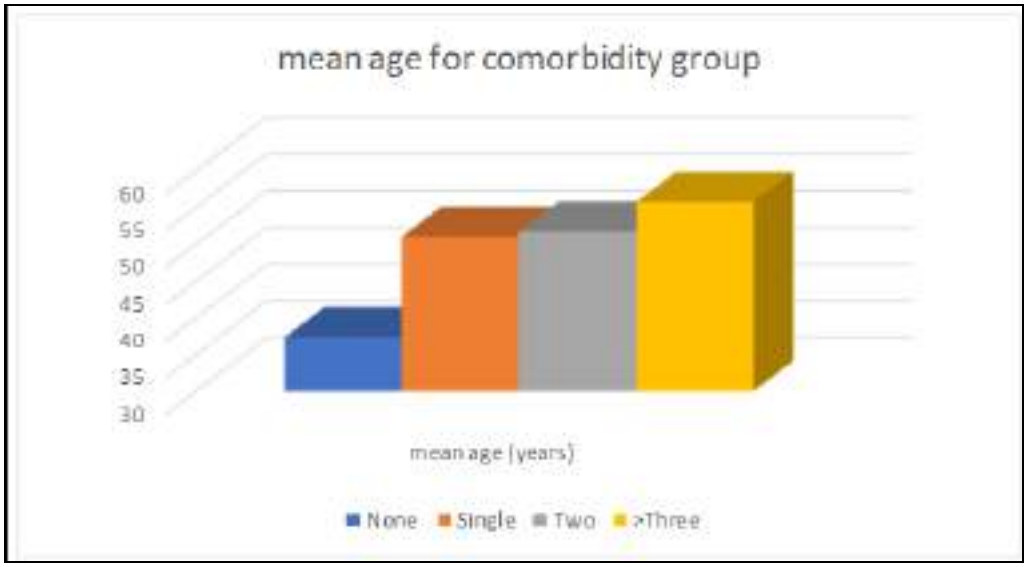


TABLE 9: PATTERN OF AKI

PATTERN	No. of cases	%
MED	289	76.1%
SUR	68	17.9%
OBS	23	6.1%

Majority of the cases of AKI were diagnosed in medical wards and ICU. 289 patients out of 31324 (9.2 cases per thousand admission) accounted for 76.1% cases of CA-AKI.

68 cases had CA-AKI in surgical group out of 16311 (4.2 cases per thousand admission) and accounted for 17.9% cases of CA-AKI.

23 cases had CA-AKI in obstetric group out of 6650 (3.45 cases per thousand admission) and accounted for 6.1% cases of CA-AKI.

Mean age of medical and surgical group was comparable (46.9 and 45.2 years) while that of obstetric group was 28.3 years.

FIGURE 10: PATTERN OF AKI

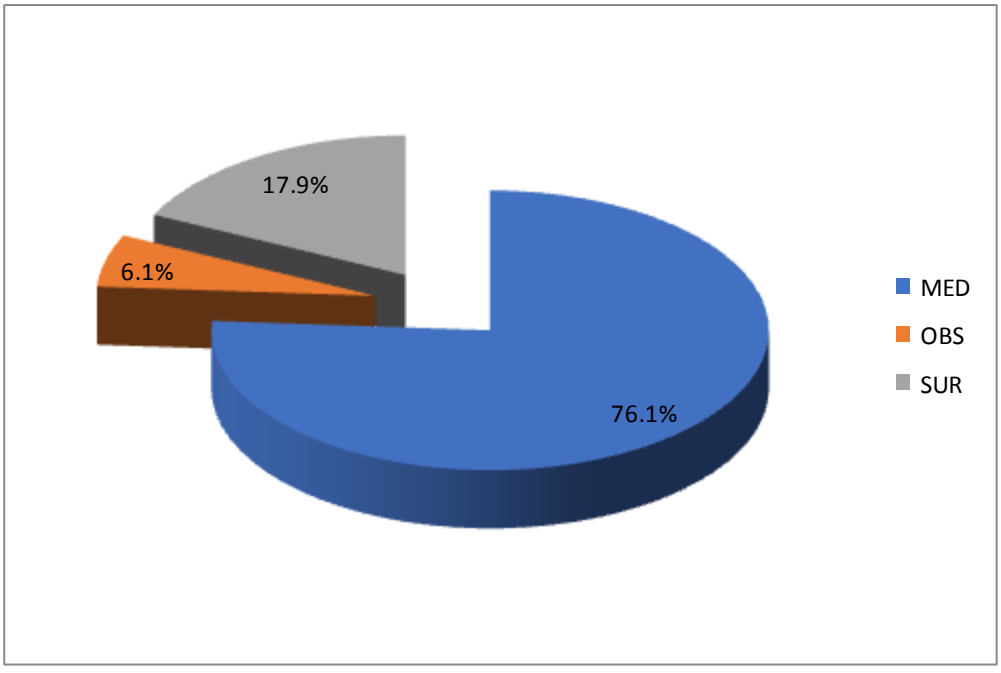


TABLE 10: MEDICAL CAUSES OF AKI

Primary Etiology of AKI	No. of cases	%
SEPSIS	77	26.6%
TROPICAL FEVER	55	19.0%
HYPOVOLEMIA	69	23.9%
DRUGS	35	12.1%
POISONING	15	5.2%
SNAKE BITE	8	2.8%
GLOMERULONEPHRITUS	8	2.8%
CARDIORENAL	15	5.2%
OTHERS (HUS, HRS, TLS, MM, G6PD)	7	2.4%

Sepsis was the most common cause of Medical-AKI (26.6 %) followed by hypovolemia (23.9%), tropical fever (19.0%), drugs (12.1%), poisoning (5.2%), cardiorenal (5.2%) snake bite 2.8% and glomerulonephritis (2.8%). HUS, HRS, TLS, G6PD and Myeloma accounted for 2.4% cases.

FIGURE 11: MEDICAL CAUSES OF AKI

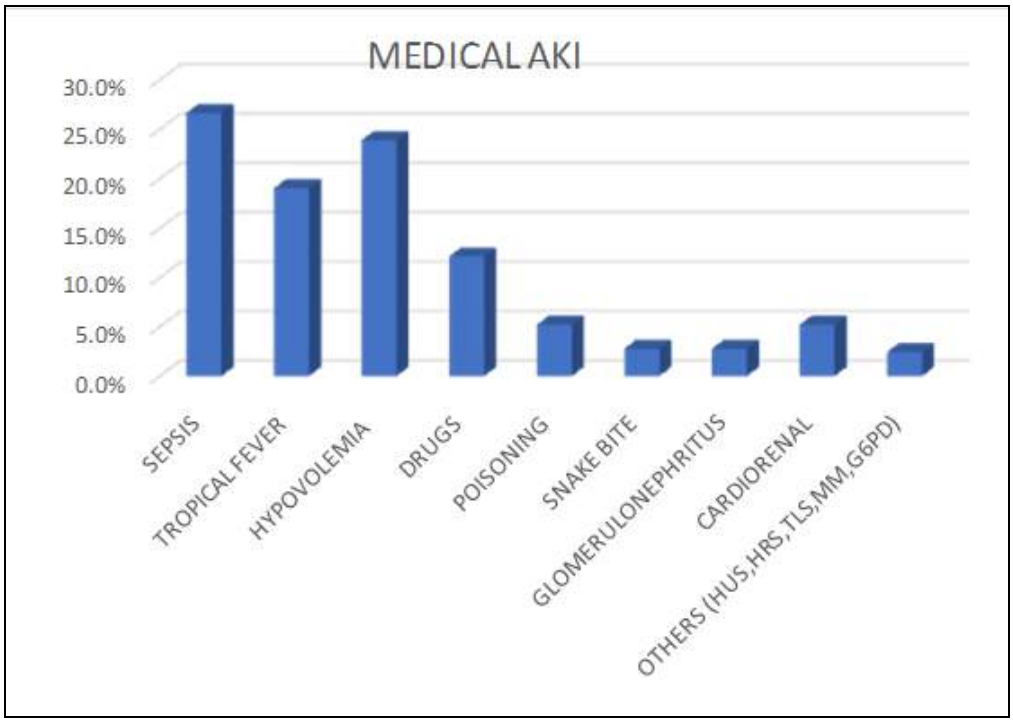


TABLE 11: SURGICAL CAUSES OF AKI

SURGICAL	No. of cases	%
NEPHROLITHIASIS	19	27.9%
CRUSH INJURY	15	22.1%
BPH	7	10.3%
D.FOOT	7	10.3%
MALIGNANCY	5	7.4%
CYSTOCELE	2	2.9%
NEUROGENIC BLADDER	1	1.5%
URETHRAL TRAUMA	3	4.4%
BURNS	2	2.9%
PAPILLARY NECROSIS	4	5.9%
PERFORATION PERITONITIS	2	2.9%
FOURNIER'S GANGRENE	1	1.5%

Urinary obstruction was the most common surgical causes of AKI (60.3%) with main etiologies being Nephrolithiasis ad BPH followed by crush injury (22.1). sepsis accounted for 17.6% cases with most common site being diabetic foot.

FIGURE 12: SURGICAL CAUSES OF AKI

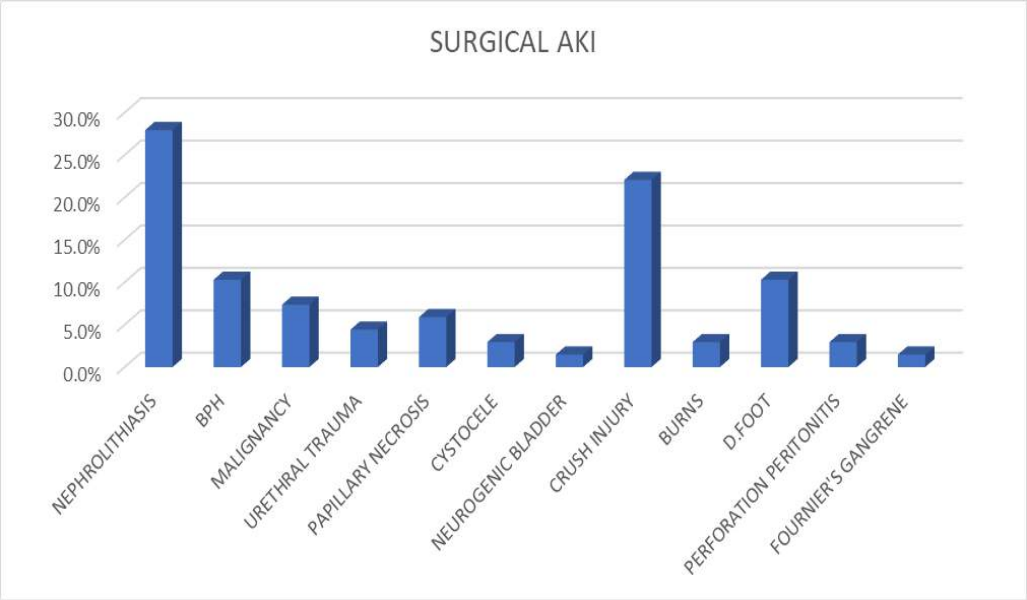


TABLE 12: OBSTETRIC CAUSES OF AKI

OBSTETRICS	No. of cases	%
PREECLAMPSIA	6	26.09%
PUERPERAL SEPSIS	9	39.13%
HUS	1	4.35%
APH	1	4.35%
PPH	3	13.04%
SEPTIC ABORTION	1	4.35%
SEPSIS/IUD	1	4.35%
HELLP	1	4.35%

48% of obstetric cases were due to sepsis, 1 case each with septic abortion and IUD. 4 were due to haemorrhage, 6 were a part of preeclampsia and 1 each due to HELLP and HUS.

FIGURE 13: OBSTETRIC CAUSES OF AKI

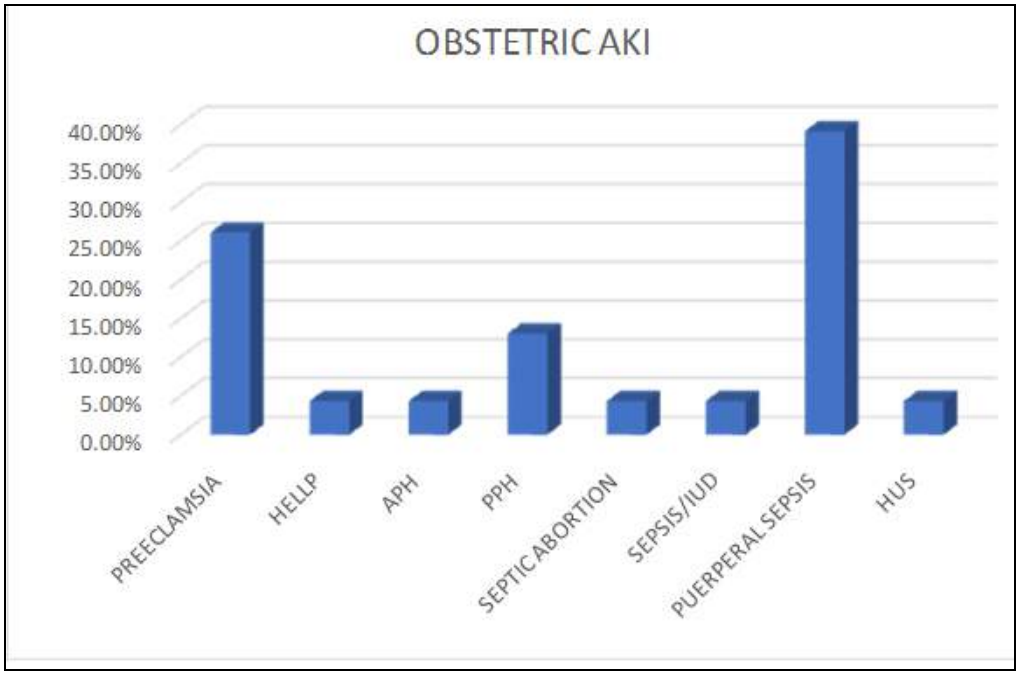


TABLE 13: ETIOLOGIES OF AKI

	No. of cases	%
INFECTIVE	155	40.3%
NONINFECTIVE	225	59.7%
	No. of cases	%
TROPICAL ETIOLOGY	203	53.4%
NON-TROPICAL	177	46.6%

155 cases (40.3%) can be attributed to infective etiologies mainly sepsis and tropical infections and rest 25(59.7%) by non-infective etiologies.

203 cases accounting for 53.4% of total cases were related to etiologies specific to tropical areas. This subgroup had mean age of 41.3 years, significantly less than the other subgroup (Non-tropical AKI with mean age 48.6 years) using an unpaired T-test with a P value less than 0.01.

FIGURE 14: ETIOLOGIES OF AKI

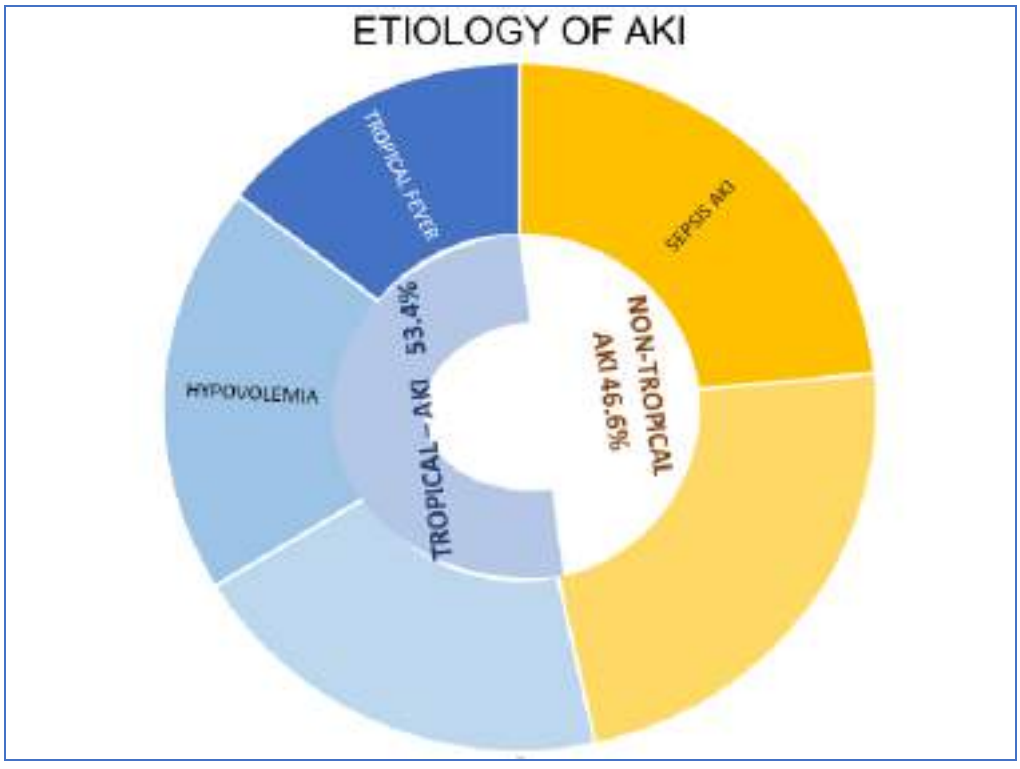


TABLE 14: INFECTIOUS CAUSES OF AKI

Etiology of AKI	No. of cases	%
SEPSIS	100	64.5%
TROPICAL FEVER	55	35.5%

A total of 100 cases of CA-AKI were due to sepsis accounting for 26.3% of total cases with a mean age of 48.2 years. 69 cases out of 100 had 1 or more comorbidity, 61 required ICU stay, 56 required renal replacement therapy. 55 cases were due to tropical fever with a mean age of 46.5 years. 26 cases out of 55 had 1 or more comorbidity, 32 required ICU stay, 14 required renal replacement therapy.

FIGURE 15: INFECTIOUS CAUSES OF AKI

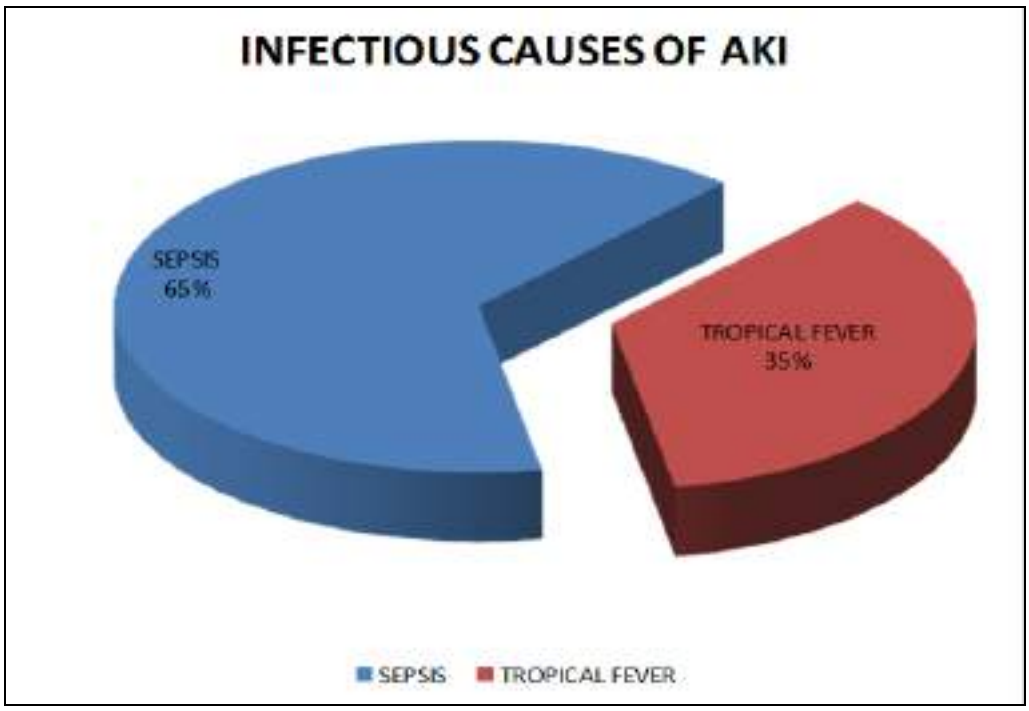


TABLE 15: SEPSIS AKI- VARIOUS ETIOLOGIES

SEPSIS	No. of cases	%
PYELONEPHRITIS	21	21%
PNEUMONIA	31	31%
GASTROENTERITIS	10	10%
PUERPERAL SEPSIS	11	11%
D.FOOT	7	7%
MENINGITIS	2	2%
INFECTIVE ENDOCARDITIS	1	1%
CHOLENGITIS	1	1%
BURNS	2	2%
SEPTIC ARTHRITIS	2	2%
COLITIS	1	1%
PERFORATION PERITONITIS	2	2%
SBP	5	5%
FOURNIER'S GANGRENE	1	1%
FOCUS NK	3	3%

Most common focus of sepsis was pneumonia (8.2% of total cases) followed by pyelonephritis (5.5% of total cases). 77% cases were medical, 12 were surgical while 11 cases were puerperal sepsis. In 3 cases focus of sepsis could not be identified.

FIGURE 16: SEPSIS AKI- VARIOUS ETIOLOGIES

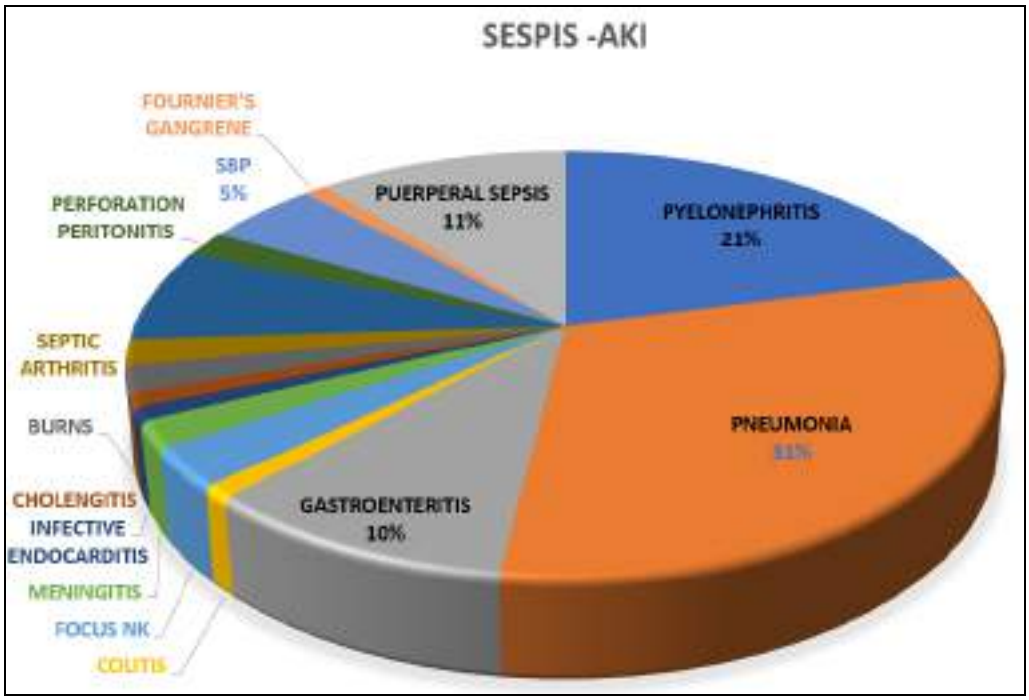


TABLE 16: VARIOUS ETIOLOGIES OF TROPICAL FEVER

TROPICAL FEVER	No. of cases	%
DENGUE	24	44%
SCRUB	12	22%
ENRETIC	9	16%
LEPTOSPIROSIS	5	9%
MALARIA	5	9%

Dengue fever was the most common tropical infection associated with AKI mainly in patients with severe dengue. Scrub typhus was the 2nd most common cause followed by enteric fever, malaria and leptospirosis.

FIGURE 17: VARIOUS ETIOLOGIES OF TROPICAL FEVER

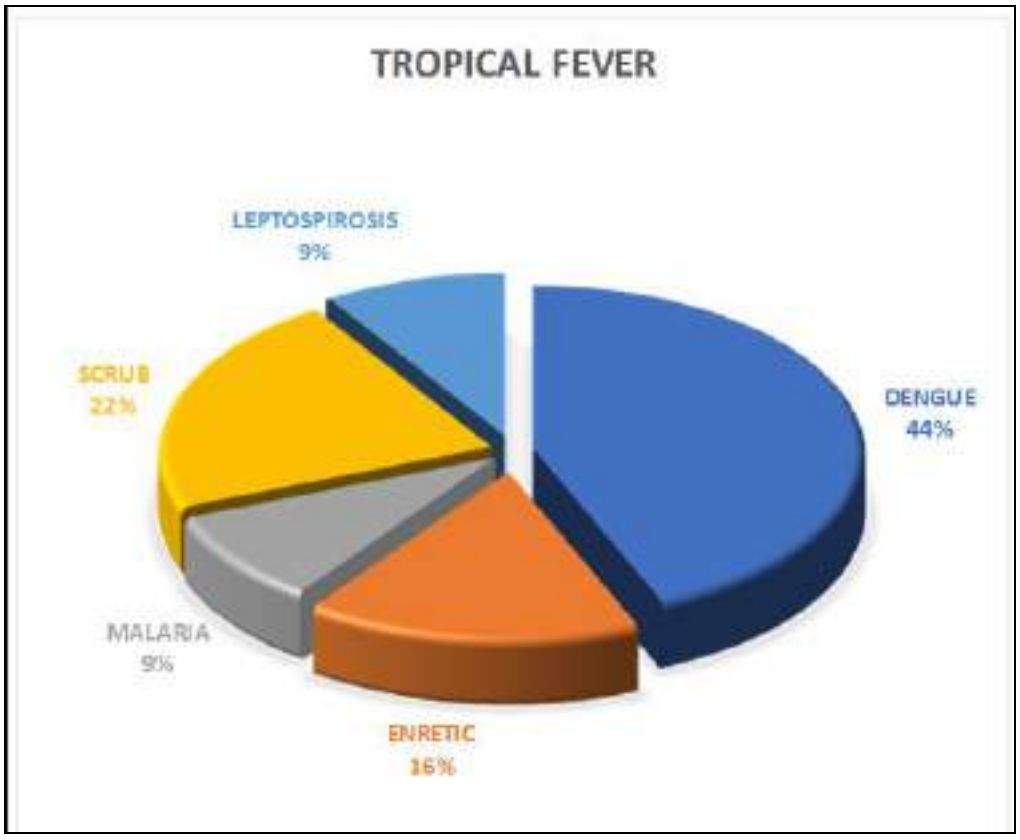


TABLE 17: NON-INFECTIVE CAUSES OF AKI

	No. of cases	%
HYPOVOLEMIA	73	32.4%
DRUGS	35	15.5%
URINARY OBSTRUCTION	41	18.2%
POISONING	15	6.7%
SNAKE BITE	8	3.5%
GLOMERULONEPHRITUS	8	3.5%
PREECLAMSIA/HELLP	7	3.1%
CARDIORENAL	15	6.7%
CRUSH INJURY	15	6.7%
OTHERS (HUS, HRS, TLS, MM, G6PD)	8	3.5%

Hypovolemia was the most common cause of CA-AKI accounting for 32.4% of cases. Their need of ICU and renal replacement therapy was 11 and 6 cases respectively and they had much shorter hospital stay in comparison to other etiologies (4.5 days vs 9.1 days overall) with a P value below 0.01 on unpaired T-test.

Obstruction accounted for 18.2% of cases followed by drugs (15.5%). There were 15 cases each of Poisoning, cardiorenal and crush injury causing traumatic rhabdomyolysis. 8 cases each were due to Vasculotoxic snake bite and glomerulonephritis. 7 cases were due to preeclampsia. There were 2 cases of HRS, 2 of HUS, 2 cases of TLS and 1 case each of Multiple myeloma and G6PD deficiency causing AKI.

FIGURE 18: NON-INFECTIVE CAUSES OF AKI

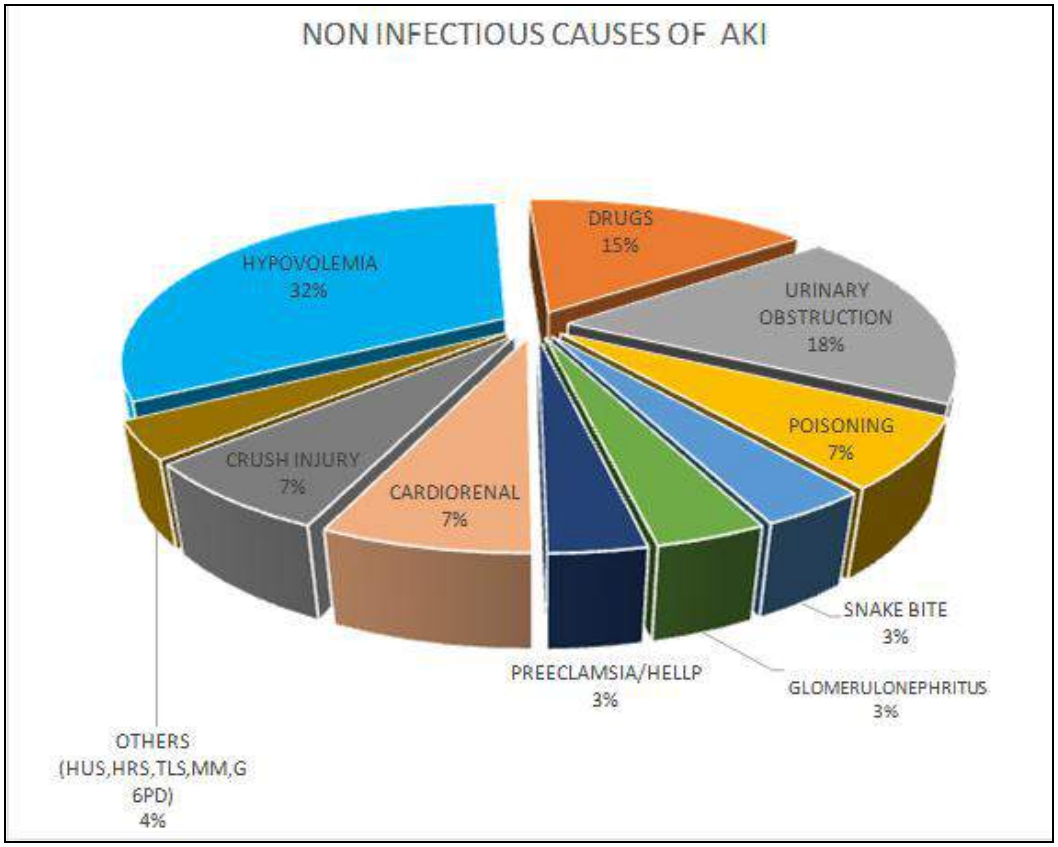
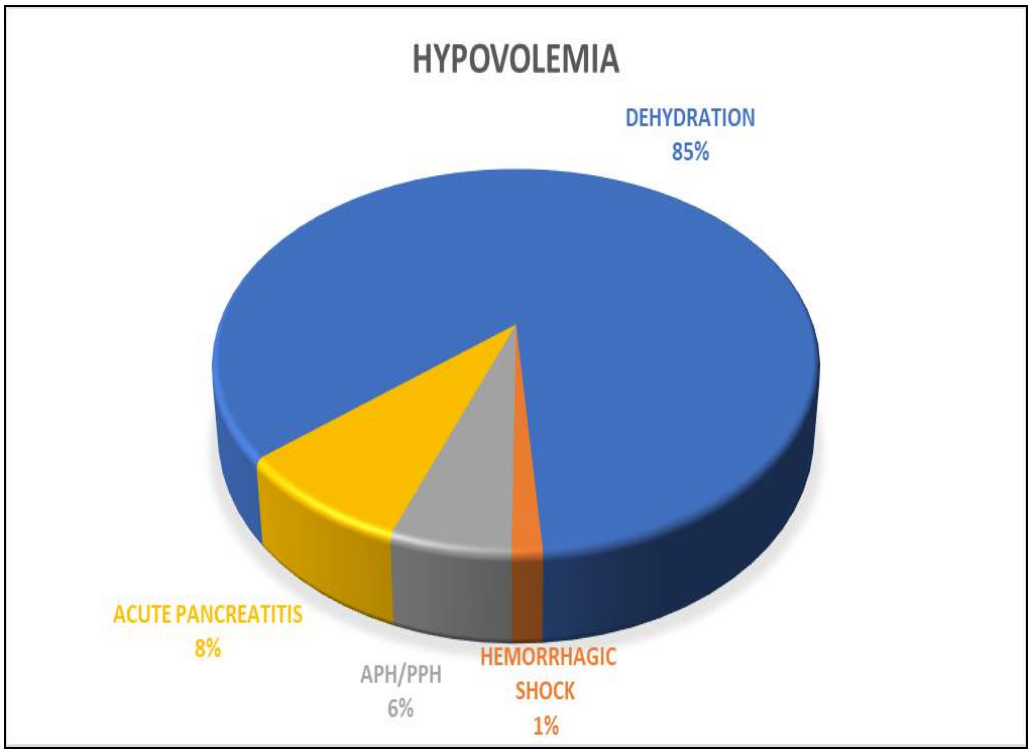


TABLE 18: VARIOUS ETIOLOGIES OF HYPOVOLEMIA

HYPOVOLEMIA	No. of cases	%
DEHYDRATION	62	85%
ACUTE PANCREATITIS	6	8%
APH/PPH	4	6%
HEMORRHAGIC SHOCK	1	1%

Dehydration was the most common cause of hypovolemia accounting for 85% of cases followed by Acute Pancreatitis, PPH/APH and Hemorrhagic shock. There were 6 cases of pancreatitis. 4 had BISAP score of 2 while 2 had BISAP score above 3.

FIGURE 19: VARIOUS ETIOLOGIES OF HYPOVOLEMIA



TABE 19: VARIOUS ETIOLOGIES OF URINARY OBSTRUCTION

URINARY OBSTRUCTION	No. of cases	% of Total
NEPHROLITHIASIS	19	46.3%
BPH	7	17%
MALIGNANCY	5	12.2%
TRAUMA	3	7.3%
U.O PAPILLARY NECROSIS	4	9.7%
CYSTOCELE	2	4.8%
NEUROGENIC BLADDER	1	2.4%

Nephrolithiasis was the most common cause of urinary obstruction either by causing bilateral obstruction, or bladder outlet obstruction or more commonly unilateral obstruction in patient with solitary functioning kidney. It was followed by BPH, urogenital malignancies, papillary necrosis, urogenital trauma, cystocele and Neurogenic bladder.

FIGURE 20: VARIOUS ETIOLOGIES OF URINARY OBSTRUCTION

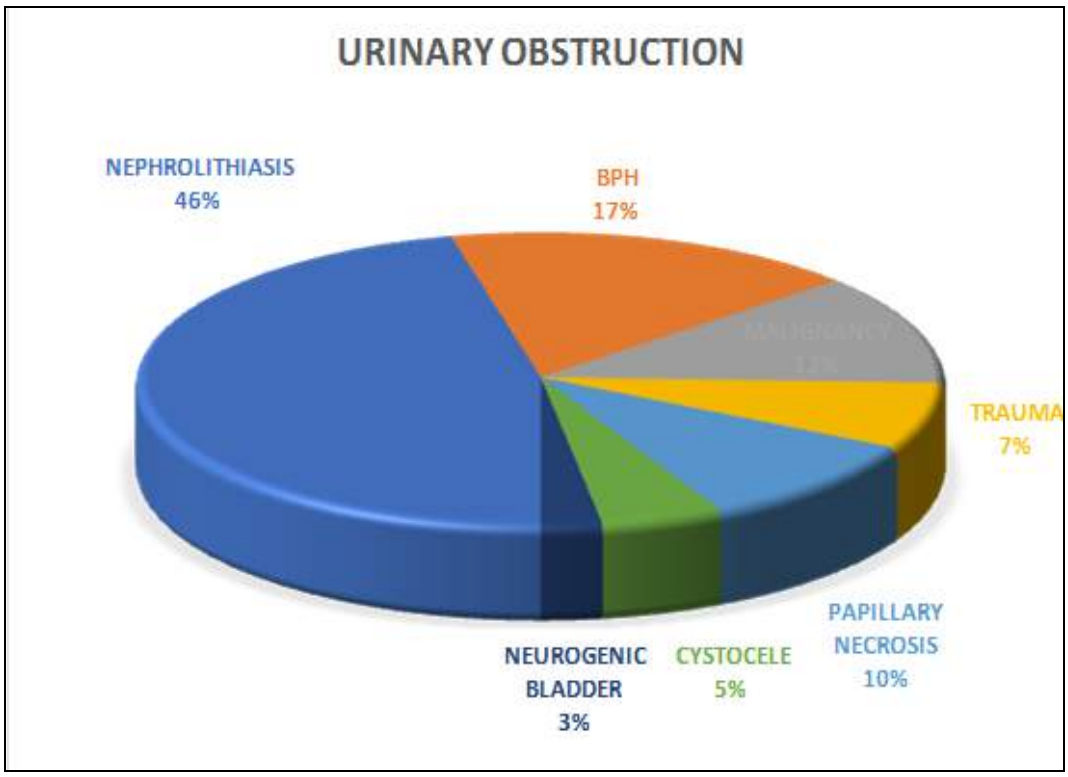


TABLE 20: VARIOUS ETIOLOGIES OF DRUG INDUCED AKI

Drugs	No. of cases	%
NSAIDS	13	37.1%
DIURETICS	6	17.1%
ACEI OR ARB'S	7	20%
CAM	3	8.5%
HYPERVITAMINOSIS D	2	5.7%
RIFAMPICIN	2	5.7%
ACYCLOVIR	1	2.8%
TENOFOVIR	1	2.8%

NSAIDS were the most common cause accounting for 37% of cases and 3.4% of total cases. It was followed by ACEI/ARB and Diuretics. CA-AKI secondary to complementary and alternative medications (CAM) was diagnosed in 3 cases. 2 patients had severe hypervitaminosis D causing hypercalcemia and AKI.

48% of these drugs were non-prescribed mainly NSAIDS and CAM. 25% cases were due to wrong dose or duration of prescribed drug while 25% cases of AKI were with regular prescription and check-up.

FIGURE 21: VARIOUS ETIOLOGIES OF DRUG INDUCED AKI

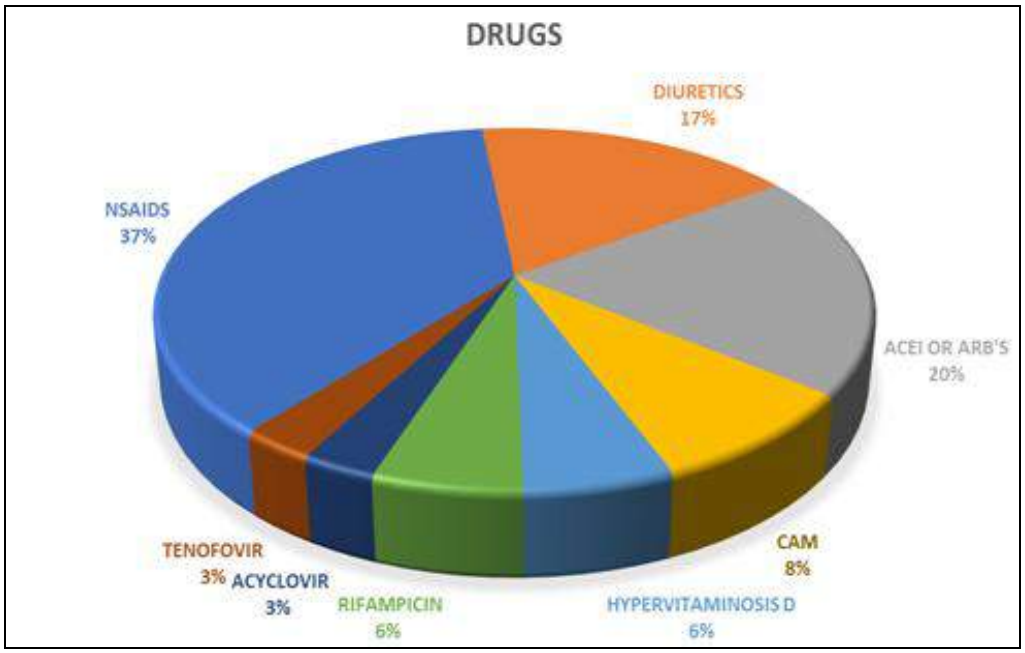
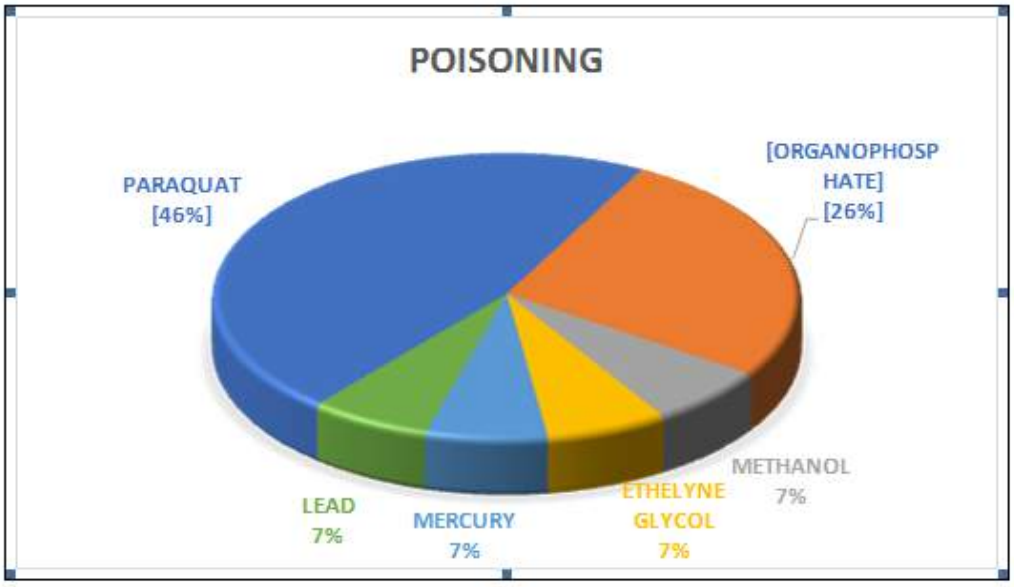


TABLE 21: VARIOUS POISONING CAUSING AKI

POISONING	No. of cases	%
PARAQUAT	7	46%
ORGANOPHOSPHATE	4	26%
METHANOL	1	7%
ETHELENE GLYCOL	1	7%
MERCURY	1	7%
LEAD	1	7%

Paraquat was the most common cause of AKI due to poisoning with total of 7 cases. Organophosphate poisoning caused 4 cases of AKI followed by one each case due to Methanol, Ethelene glycol, mercury and lead. Majority of the cases were suicidal (11 cases) followed by accidental (3 cases). Lead poisoning was due to industrial exposure.

FIGURE 22: VARIOUS POISONING CAUSING AKI



There were at least 57 cases, where more than one etiology was responsible for CA-AKI accounting of 15% of total cases. Most of these cases (40 cases) had sepsis as the primary etiology with second etiology either being nephrotoxic drugs, hypovolemia, cardiac dysfunction or urinary obstruction.

In 6 patients, an etiological diagnosis could not be reached and renal biopsy was either not feasible or not consented. 3 of these patients were suspected to have some tropical infection while the other 3 were suspected to have intake of nephrotoxic medications.

TABLE 22: NEED OF ICU STAY

Need of ICU	No. of cases	%
Yes	159	41.8%
No	221	58.2%

A Total of 159 patients (41.8%) patients required ICU stay with average ICU stay of 6.8 days and average hospital stay of 9.1 days.

FIGURE 23: NEED OF ICU STAY

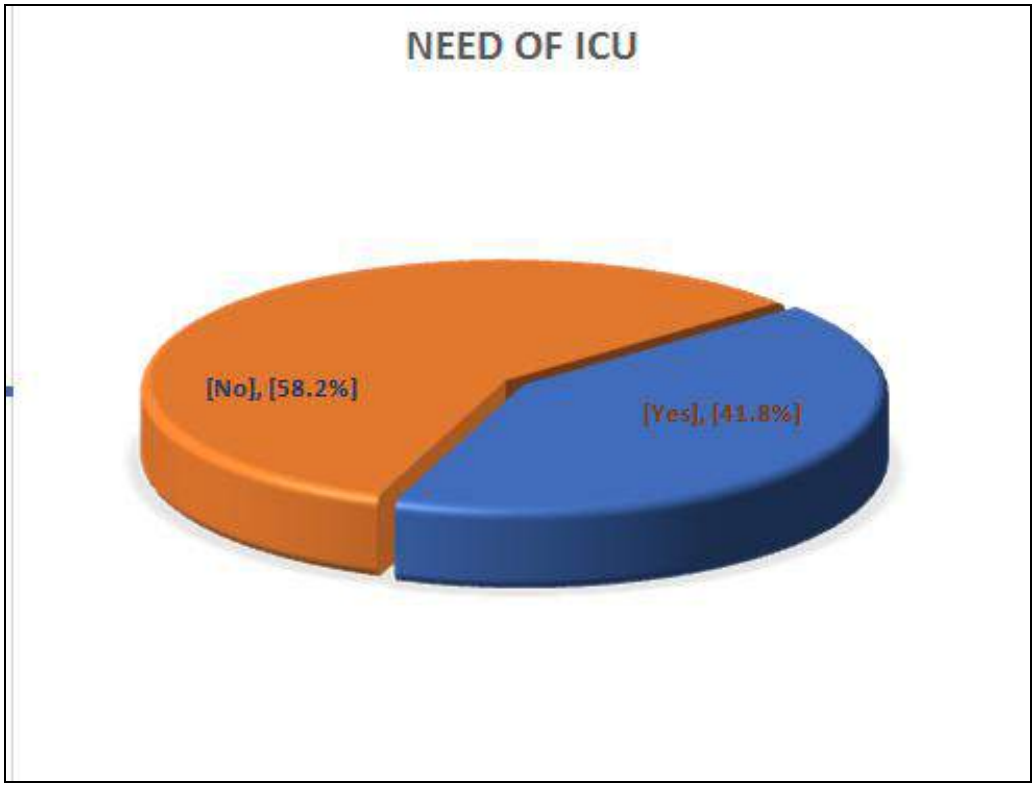


TABLE 23: AVERAGE ICU AND HOSPITAL STAY

	Mean	SD
Average hospital stay	9.1	5.04
Average ICU stay	6.8	4.47

FIGURE 24: AVERAGE ICU AND HOSPITAL STAY

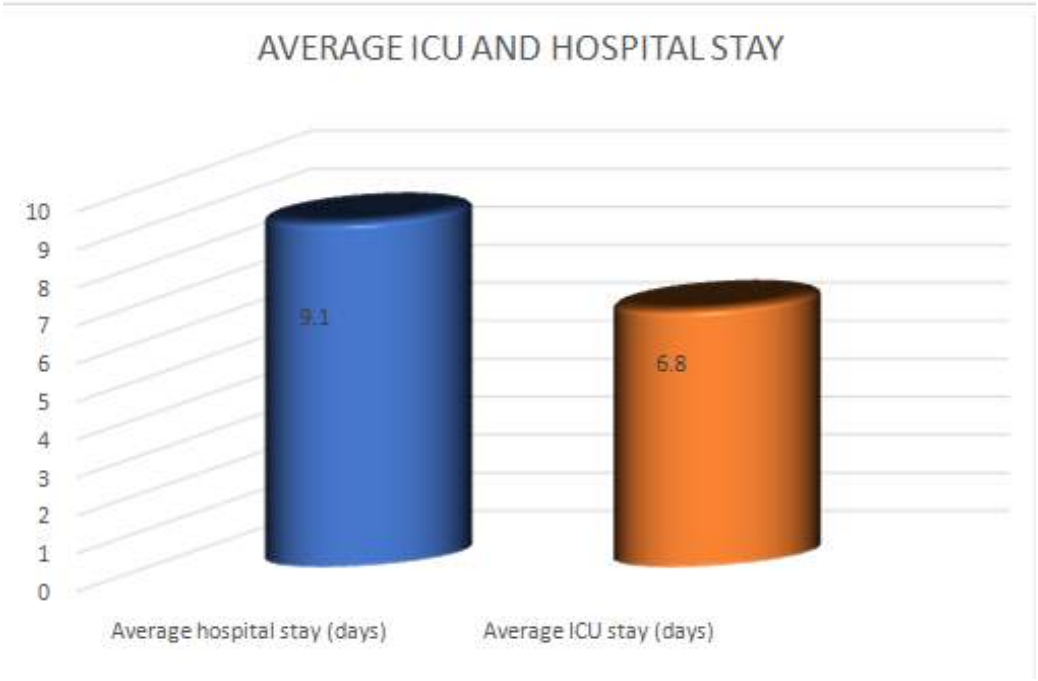


TABLE 24: TYPE OF AKI

	No. of cases	%
RENAL HYPOPERFUSION AT ONSET	158	41.6%
INTRINSIC RENAL	181	47.6%
OBSTRUCTIVE (POST-RENAL)	41	10.8%

47.6% cases of AKI were classified as intrinsic AKI and it was the most common type of AKI. It was followed by 41.6% cases of Pre-renal (Renal hypoperfusion to begin with). 41 cases accounting for 10.8% cases were due to urinary obstruction (Post-renal).

FIGURE 25: TYPE OF AKI

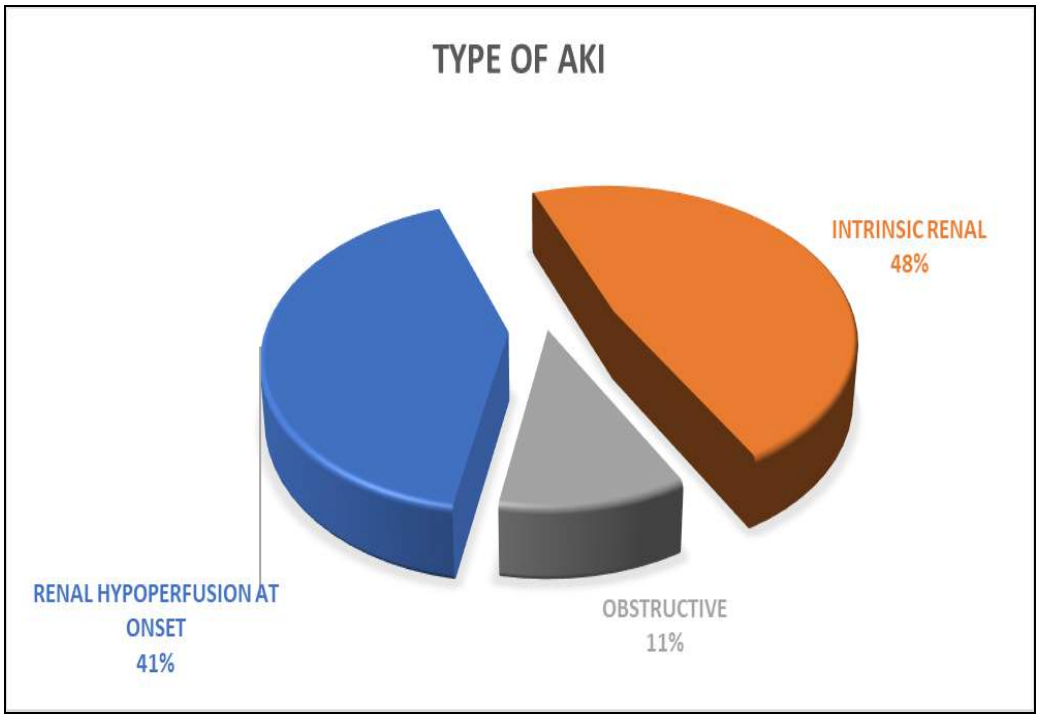


TABLE 25: STAGING OF AKI

KDIGO STAGE OF AKI	No. of cases	%
1	26	6.8%
2	139	36.6%
3	215	56.6%
Total	380	100.0%

Most of the patients were having stage 3 AKI with 56.6% cases followed by stage 2 AKI accounting for 36.6% cases. Only 6.8% cases were diagnosed as stage 1 AKI. None of the patient with stage 1 AKI required ICU stay, had average hospital stay of 3.7 days, and none required readmission over 1 month. In patients with Stage 2 AKI, 25(18%) required ICU with average ICU stay of 4.2 days and average hospital stay of 7.1 day. 2 required readmission within one month of discharge. In patients with stage 3 AKI, 134 patients (62%) required ICU stay with average ICU stay of 7.3 days and average hospital stay of 11 days. On comparing duration of ICU stay as well as hospital stay between these 3 groups, the difference was statistically significant between the 3-group using one-way ANOVA with P values below 0.05.

FIGURE 26: STAGING OF AKI

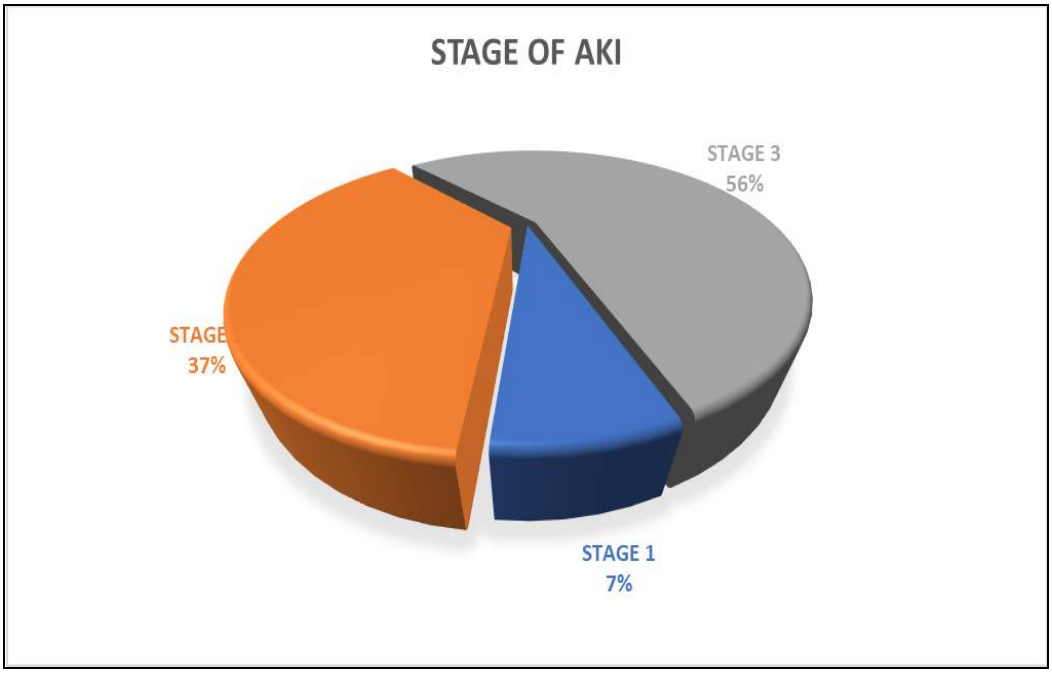


TABLE 26: NEED OF RENAL REPLACEMENT THERAPY

RENAL REPLACEMENT THERAPY	No. of cases	%
Yes	123	32.4%
No	257	67.6%
Total	380	100.0%

A total of 123 patients (32.4%) patients required Renal replacement therapy (R.R.T.). The criteria for starting renal replacement therapy and for deciding mode of renal replacement therapy were not uniform and varied between different treating nephrologist. Mostly traditional indications of refractory hyperkalaemia, metabolic acidosis, fluid overload or features of uraemia were used to guide need of R.R.T.

FIGURE 27: NEED OF RENAL REPLACEMENT THERAPY

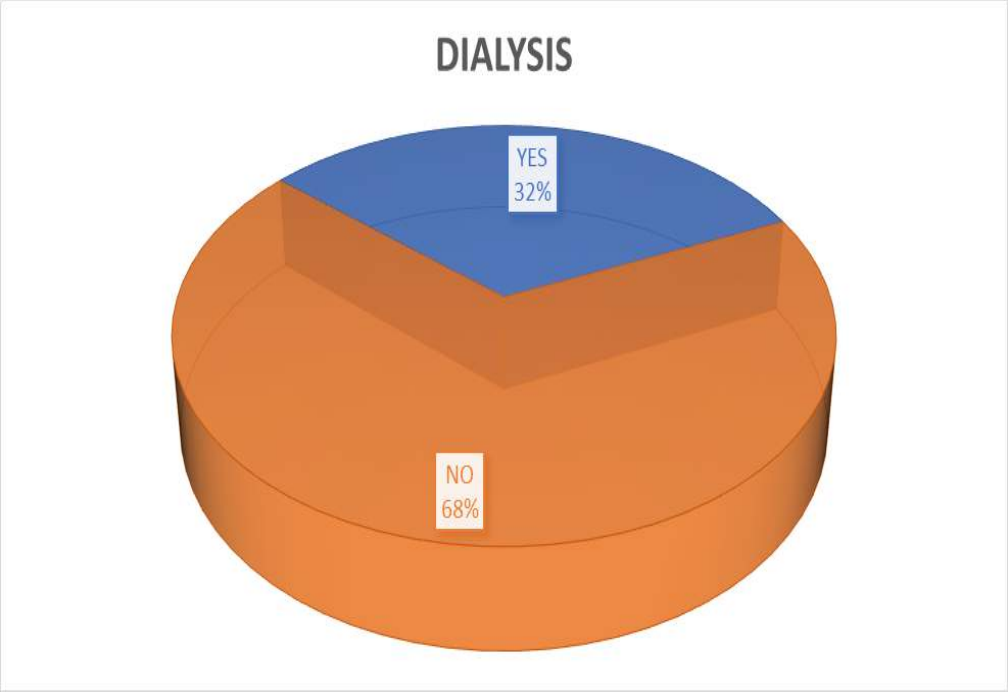


TABLE 27: VARIOUS DIALYTIC MODALITIES

Initial dialysis modality	No. of cases	%
HD	50	40.7%
SLED	37	30.1%
PD	28	22.8%
CRRT	8	6.5%
Total	123	100.0%

Most common dialytic modality used was hemodialysis used in 50 cases (40.7%). It was used in hemodynamically stable patients with 52% patients not requiring ICU care. Rest all modality of dialysis were performed in ICU setting in hemodynamically unstable patients, most commonly Sustained low efficiency dialysis (SLED) followed by peritoneal dialysis (PD) and CRRT. The choice of modality varied with different Nephrologist and SLED was the most preferred modality in hemodynamically unstable patients.

FIGURE 28: VARIOUS DIALYTIC MODALITIES

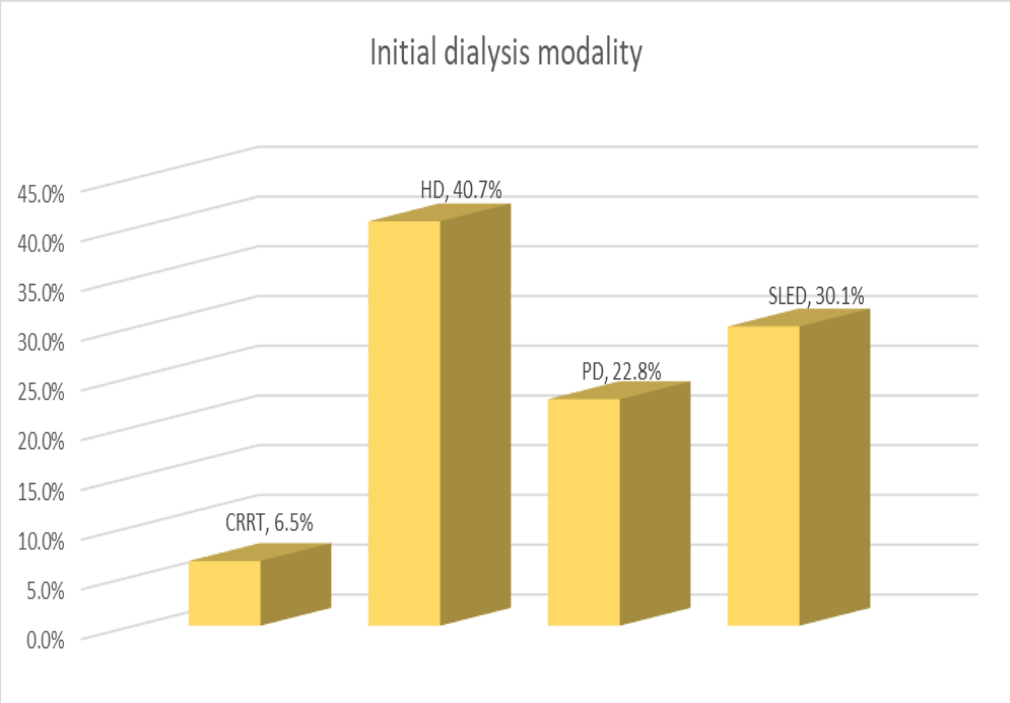


TABLE 28: RENAL STATUS AT DISCHARGE AND AT 1 MONTH

Outcome	Outcome at discharge		Outcome at 1 month	
	No. of cases	%	No. of cases	%
Complete Recovery (CR)	76	20.0%	173	45.5%
Partial Recovery (PR)	179	47.1%	103	27.1%
No Recovery (NR)	63	16.6%	31	8.2%
DEATH	62	16.3%	73	19.2%
Total	380	100.0%	380	100.0%

A total of 76 patients completely recovered till the time of discharge while at the end of one month 97 more patients recovered completely making a total of 173 patients (45.5%).

179 patients had partial recovery till the time of discharge which decreased to 103 (27.1%) cases by the end of 1 month.

63 patients had no recovery at time of discharge out of which 32 patients by one month were either partially or completely recovered leaving 31(8.2%) patients with no recovery by 1 month.

62 patients died during hospital stay. This include those patients also who took discharge in a moribund state and telephonically confirmed to have died. 11 patients died during readmission within 1 month of discharge.

31 patients classified as partial recovery were on evaluation had evidence of underlying chronic kidney disease but didn't had baseline creatinine to segregate them correctly into partial vs complete recovery from this episode of CA-AKI.

FIGURE 29: RENAL STATUS AT DISCHARGE AND AT 1 MONTH

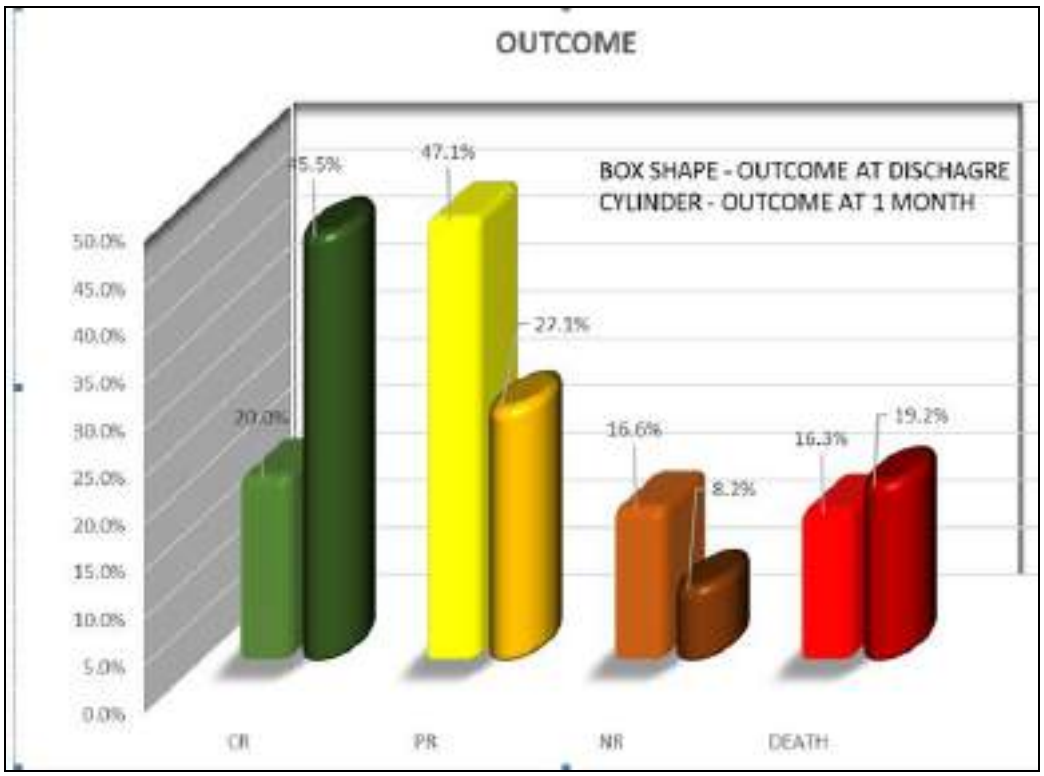


TABLE 29: 7-DAY AND 30-DAY MORTALITY

Mortality	No. of cases	%
<7 DAY	41	56.2%
7-30 DAY	32	43.8%
30 DAY	73	100.0%

7-day mortality was 41 accounting for 10.8% of all cases and 56% of all mortality cases. 32 cases died between 7 to 30 days post admission including 11 cases who died on readmission.

FIGURE 30: 7-DAY AND 30-DAY MORTALITY

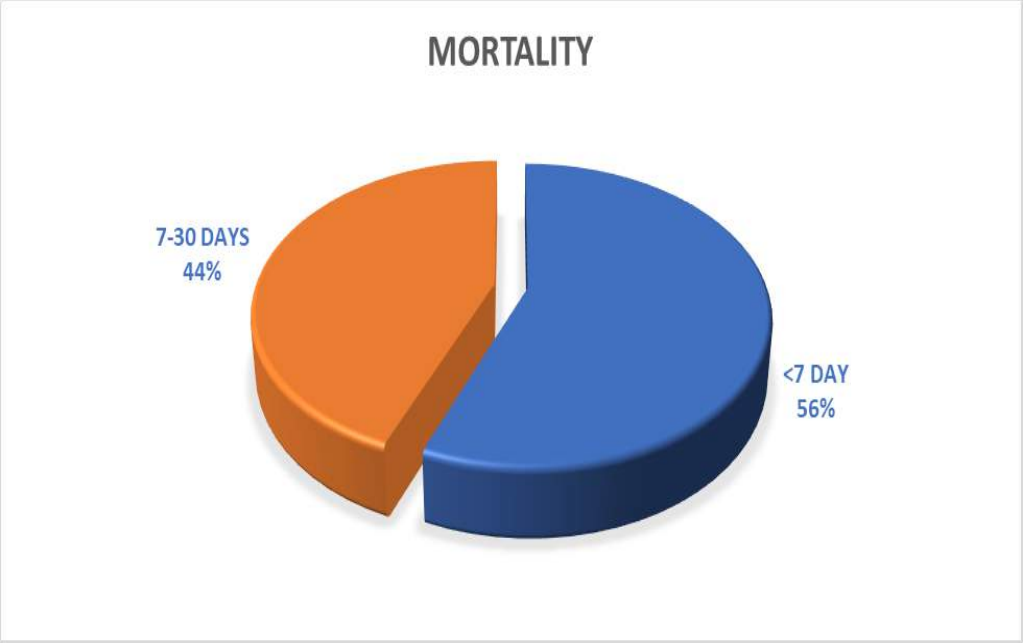


TABLE 30: ETIOLOGIES OF MORTALITY AT 7 DAYS AND 30 DAYS

Mortality	7-day Mortality		30-day Mortality		
	No. of cases	%	No. of cases	%	% per disease
SEPSIS	27	65.9%	30	41.1%	30%
PUERPERAL SEPSIS	4	9.8%	4	5.5%	36%
TROPICAL FEVER	6	14.6%	13	17.8%	23.6%
HYPOVOLUMIA	1	2.4%	1	1.4%	1%
PANCREATITIS	0	0.0%	1	1.4%	1%
DRUGS	1	2.4%	6	8.2%	17%
HRS	2	4.9%	2	2.7%	100%
POISONING	0	0.0%	9	12.3%	60%
SNAKE BITE	0	0.0%	2	2.7%	25%
CRUSH INJURY	0	0.0%	3	4.1%	20%
CARDIRENAL	0	0.0%	2	2.7%	13.3%

Sepsis was the most common cause of 7- day mortality as well as overall 30-day mortality. 31 patients (76%) out of 41 in 7-day mortality were due sepsis including puerperal sepsis. Overall 34% patient with sepsis-CA-AKI succumbed to their illness. It was followed by tropical fever with 23.6% patients dying due to it accounting for 17.8% cases of overall mortality. Poisoning and drugs mainly caused mortality between 7-30 days. Poisoning as well as HRS had very high rate of mortality 60% and 100% respectively. 1 patient died due to acute pancreatitis with BISAP score of 4.

FIGURE 31: ETIOLOGIES OF MORTALITY AT 7 DAYS AND 30 DAYS

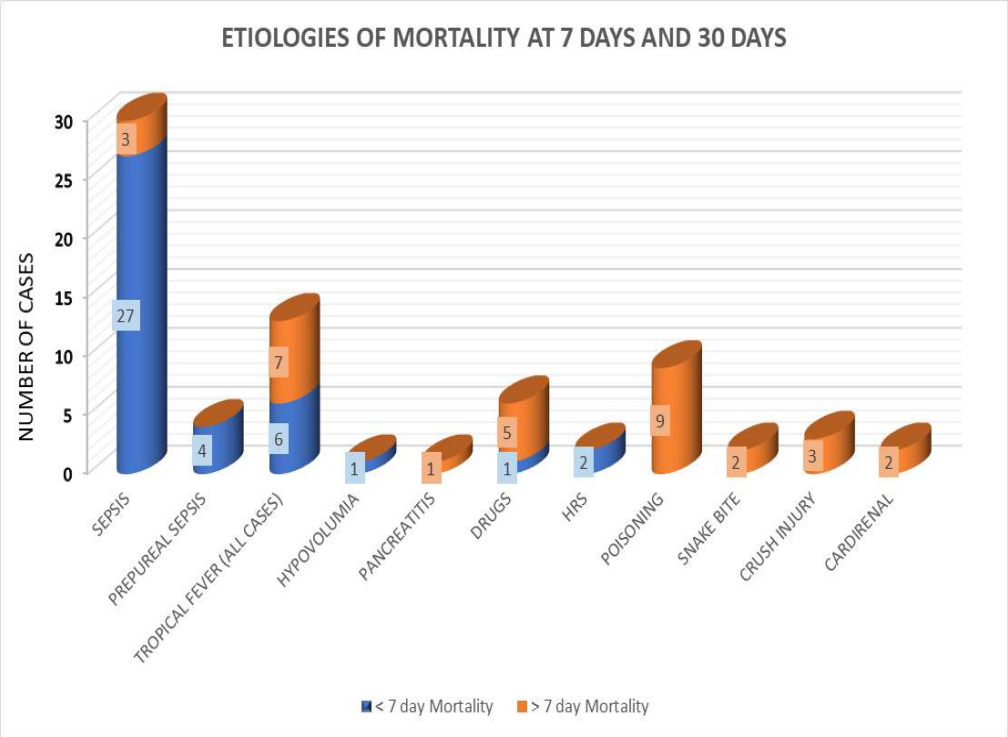


TABLE 31: MORTALITY RATE OF VARIOUS COMORBIDITIES

30 DAY MORTALITY	NO. OF CASES	% CASES	% PER DISEASE
None	25	34.25	15.34
DM	20	27.40	32.79
HTN	33	45.21	27.73
CKD	36	49.32	33.96
CAD	6	8.22	21.43
CVA	7	9.59	36.84
COAD	5	6.85	22.73
CLD	13	17.81	43.33
OTHERS	9	12.33	16.98

Patients without any comorbidities accounted for 34 % case of mortality and had 15% mortality for its subgroup. Diabetic patients had 32.8% overall mortality and accounted for 27% of mortality cases. Hypertensives had 27.7% mortality with 45% cases of mortality being hypertensive. CKD population had 34% mortality. It was 21% for patients with CAD, 36% for CVA, 43% for CLD and 22% for COAD.

FIGURE 32: MORTALITY RATE OF VARIOUS COMORBIDITIES

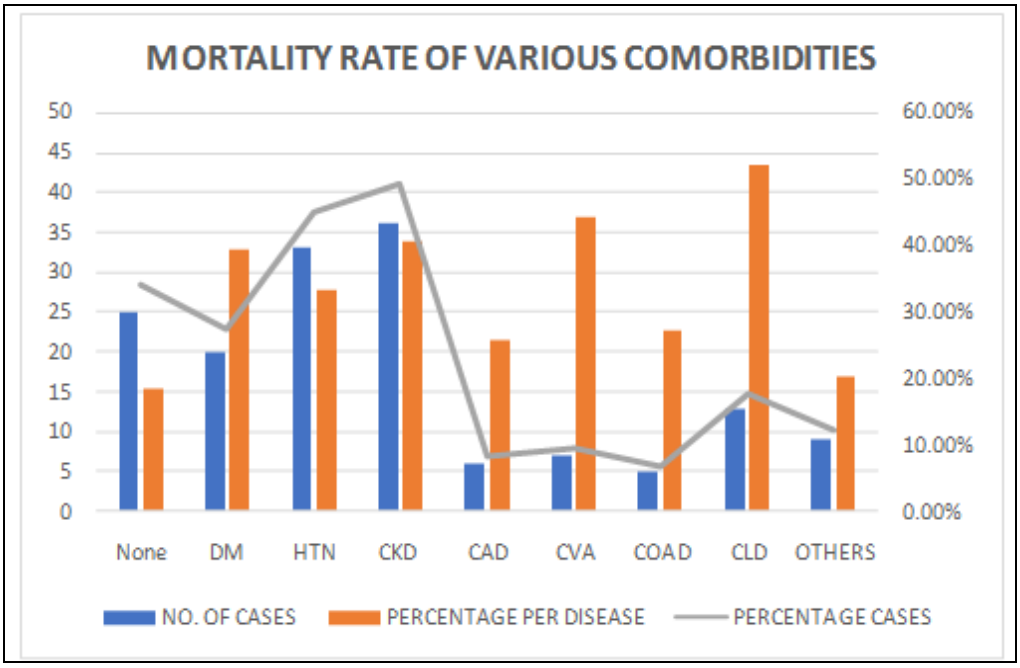


TABLE 32: MORTALITY AS PER NUMBER OF COMORBIDITIES

NO. OF COMORBIDITIES	30 DAY MORTALITY	% PER GROUP
NONE	25	15.43
1	11	10.28
2	20	26.67
> 3	17	48.57

CA-AKI patients without any comorbidity had 15.4% chance of dying due to illness while with 1 comorbidity it was 10.2%, with 2 comorbidities it was 26.6% and 48% with those having 3 or more comorbidities.

FIGURE 33: MORTALITY AS PER NUMBER OF COMORBIDITIES

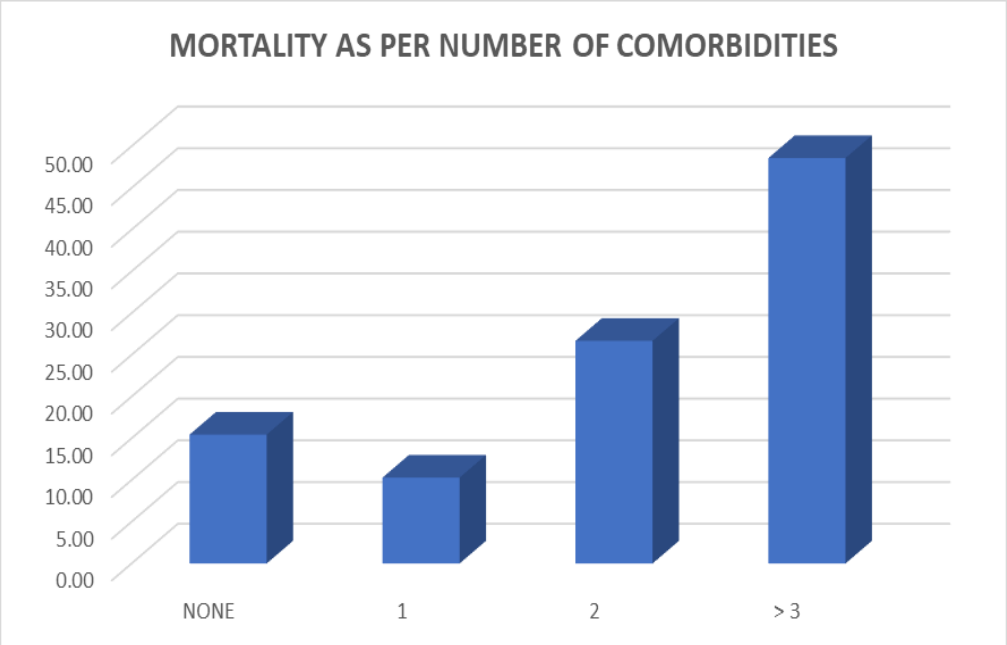


TABLE 33: PATIENT REQUIRING READMISSION WITHIN 1 MONTH

Readmission within 1 month	No. of cases	%
YES	96	30.2%
NO	222	69.8%
Total	318	100.0%

96 patients i.e. 30.2% required readmission within one month of discharge. 11 out of 96 patients died during readmission

FIGURE 34: PATIENT REQUIRING READMISSION WITHIN 1 MONTH

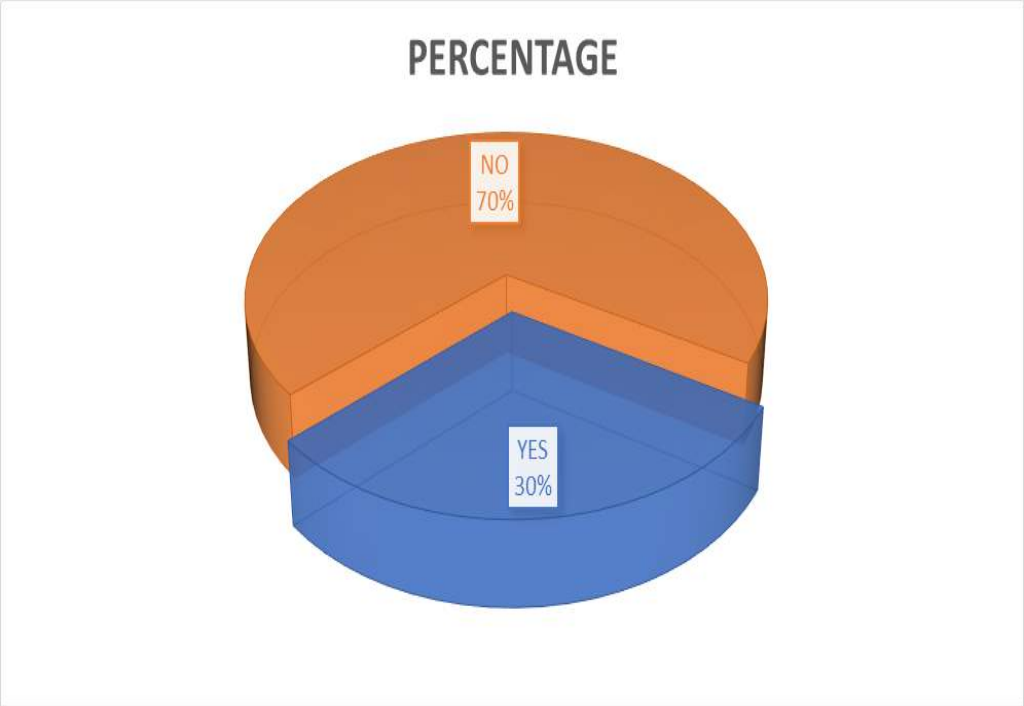


TABLE 34: ETIOLOGY OF AKI IN PATIENT REQUIRING READMISSION

Readmission within 1 month	No. of cases	% OF CASES	% PER DISEASE
SEPSIS	26	27.08%	26%
URINARY OBSTRUCTION	16	16.67%	39%
DRUGS	12	12.50%	34%
TROPICAL FEVER (ALL CASES)	10	10.42%	18%
CARDIRENAL	6	6.25%	40%
PANCREATITIS	4	4.17%	50%
GLOMERULONEPHRITIS	4	4.17%	50%
OBSTETRIC CASES	4	4.17%	17%
SNAKE BITE	5	5.21%	62.5%
CRUSH INJURY	5	5.21%	33%
POISONING	1	1.04%	2.8%
OTHER	3	3.13%	42%

Sepsis-CA-AKI patients accounted for maximum number of readmissions with 27% cases. Patients with urinary obstruction and drugs also had high rate of readmission with 39% and 34% cases respectively requiring readmission accounting for 16.7% and 12.5% of readmissions. Patients with pancreatitis, glomerulonephritis, snake bite and cardiorenal syndrome also had high rate of readmission.

FIGURE 35: ETIOLOGY OF AKI IN PATIENT REQUIRING READMISSION

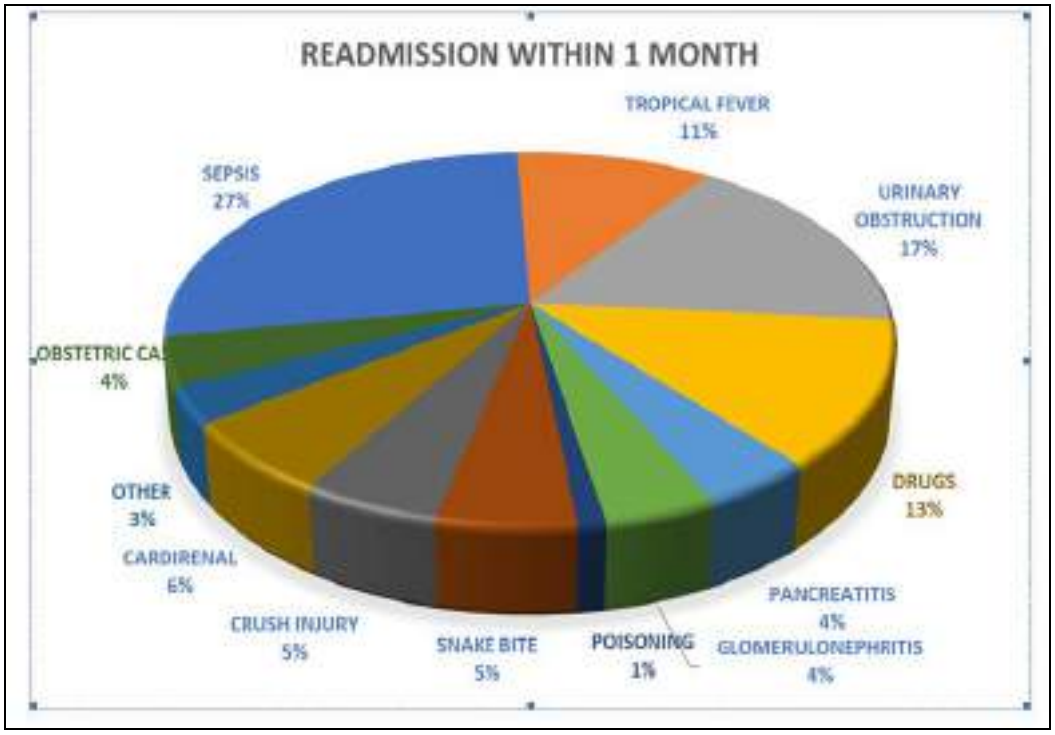


TABLE 35: READMISSION RATE FOR VARIOUS COMORBIDITIES

COMORBIDITIES	Readmission within 1 month	% PER DISEASE	% CASES
None	27	16.67	28.13%
DM	26	42.62	27.08%
HTN	29	24.37	30.21%
CKD	42	39.62	43.75%
CAD	13	46.43	13.54%
CVA	8	42.11	8.33%
COAD	9	40.91	9.38%
CLD	9	30.00	9.38%
OTHERS	16	30.19	16.67%

Of the readmitted patients, 43.7% had CKD, 30 % were hypertensive, 27% were diabetic, 13% had CAD, and ~9% had CVA, COAD and CLD each.

FIGURE 36: READMISSION RATE FOR VARIOUS COMORBIDITIES

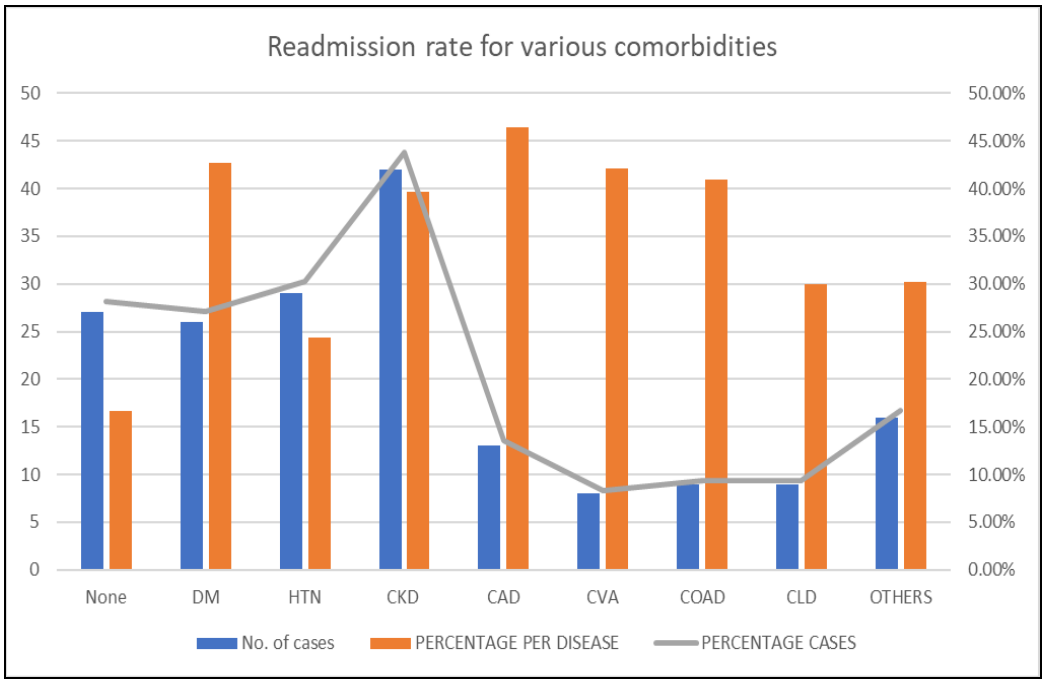


TABLE 36: READMISSION RATE AS PER NUMBER OF COMORBIDITIES

NUMBER OF COMORBIDITIES	Readmission within 1 month	% PER GROUP
NONE	27	16.67
1	27	25.23
2	29	38.67
> 3	15	42.86

Readmission rate for patients without comorbidity was 16.7% which was 25% for patient with 1 comorbidity, 38% for 2 comorbidity and 43% for patients with 3 or more comorbidity.

FIGURE 37: READMISSION RATE AS PER NUMBER OF COMORBIDITIES

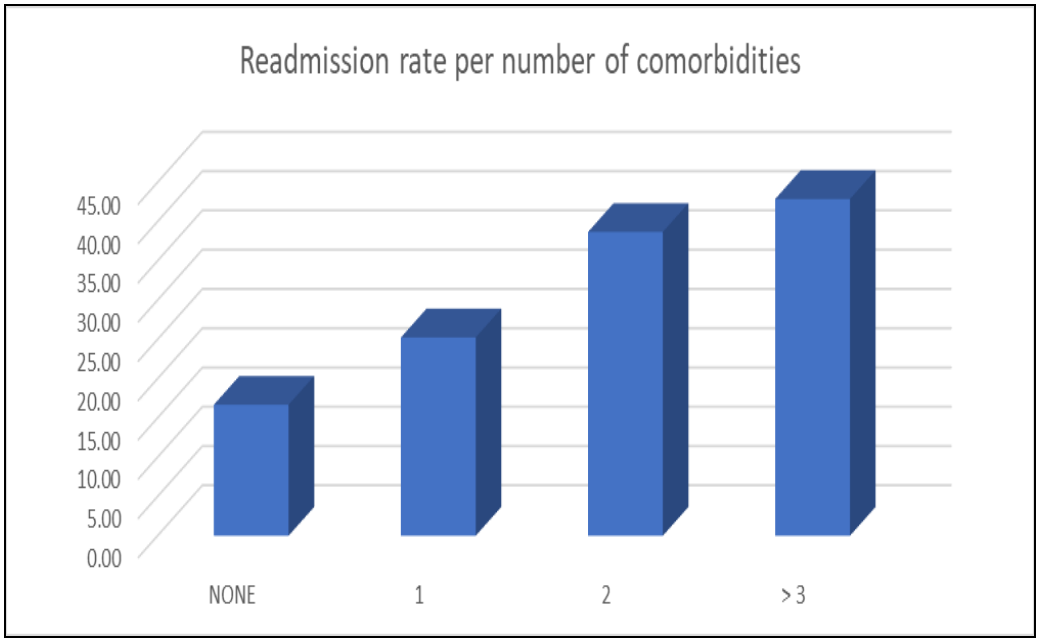


TABLE 37: PATIENT REQUIRING RENAL BIOPSY

INDICATION	ADVISED RENAL BIOPSY	PERFORMED BIOPSY
UNEXPLAINED AKI	16	10
PROLONGED AKI	19	6
GLOMERULAR DISEASE	10	10

A total of 45 patients who presented as CA-AKI were advised renal biopsy, out of which 26 renal biopsies were performed. Rest were not performed mainly due to refusal of consent in 16 cases and non-feasibility in 3 cases. 16 cases had unexplained AKI while 19 renal biopsies were advised due to prolonged non recovery. 10 patients had suspected glomerular disease.

FIGURE 38: PATIENT REQUIRING RENAL BIOPSY

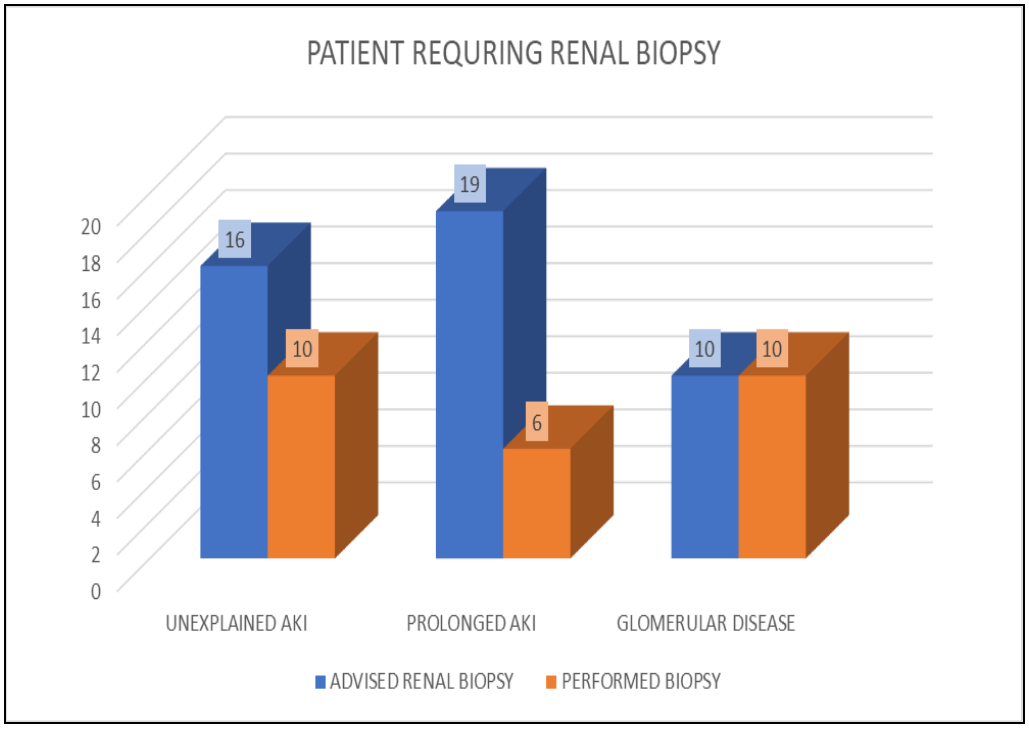
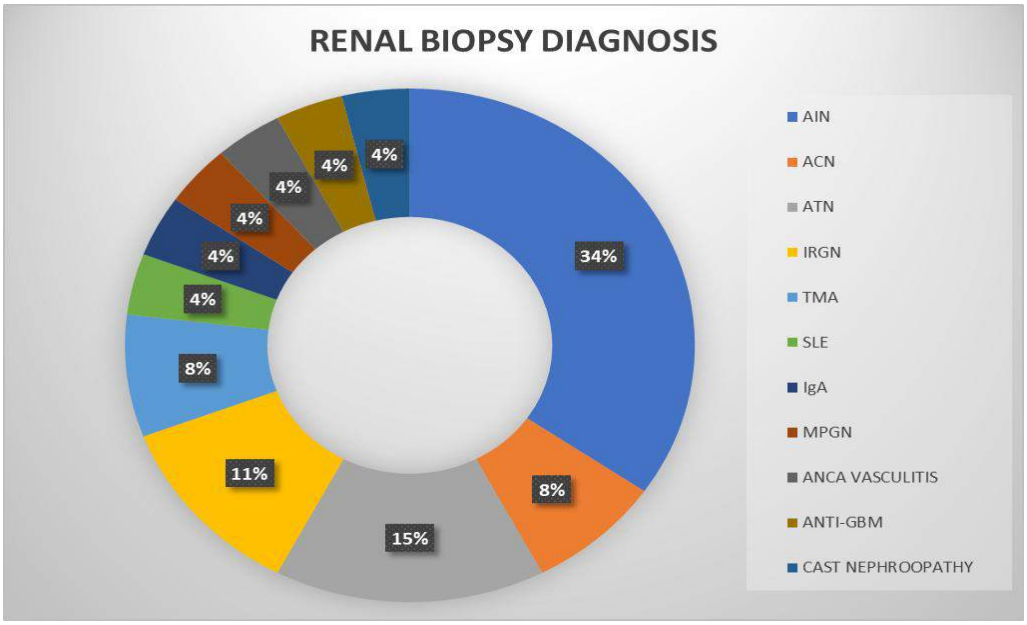


TABLE 38: RENAL BIOPSY DIAGNOSIS

RENAL BIOPSY DIAGNOSIS	No. of cases	%
AIN	9	34.6%
ACN	2	7.7%
ATN	4	15.4%
IRGN	3	11.5%
TMA	2	7.7%
SLE	1	3.8%
IgA	1	3.8%
MPGN	1	3.8%
ANCA VASCULITIS	1	3.8%
ANTI-GBM	1	3.8%
CAST NEPHROOPATHY	1	3.8%

Acute Interstitial Nephritis was the most common histological diagnosis (34%), followed by ATN (15.4%). Infection related glomerulonephritis was present in 11.5% cases followed by acute cortical necrosis and thrombotic microangiopathy accounting for 7.7% cases each. There was 1 case each of SLE, IgA nephropathy, MPGN, ANCA vasculitis, Anti-GBM disease and Cast nephropathy.

FIGURE 39: RENAL BIOPSY DIAGNOSIS



COMPARISON OF SURVIVAL AND NON-SURVIVAL GROUP

TABLE 39: COMPARING AGE, HOSPITAL STAY AND ICU STAY OF SURVIVOR VS NON-SURVIVOR

	SURVIVOR		NON-SURVIVOR		p-value
NUMBER	307		73		
	Mean	SD	Mean	SD	
Age years	45.44	11.76	45.44	12.18	0.99
Hospital stay days	9.14	4.80	8.89	6.00	0.70
ICU Stay days	6.90	4.20	6.70	4.80	0.53

There was no difference between survivor and non- survivor with respect to age, hospital stay or ICU stay.

FIGURE 40: COMPARING AGE OF SURVIVOR VS NON-SURVIVOR

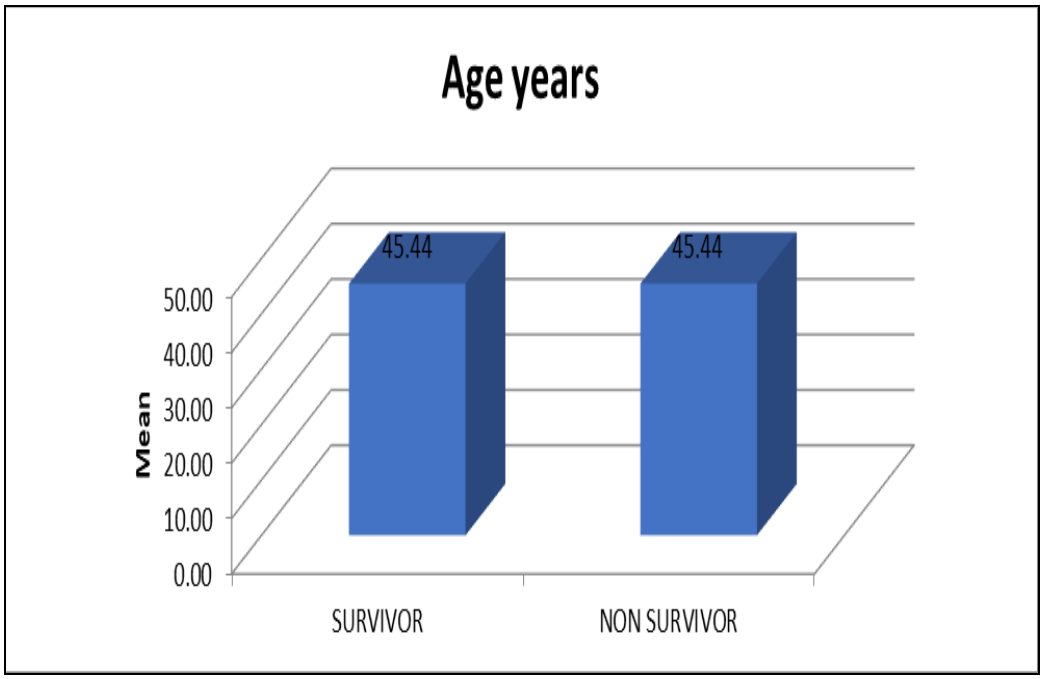


FIGURE 41: COMPARING HOSPITAL STAY OF SURVIVOR VS NON-SURVIVOR

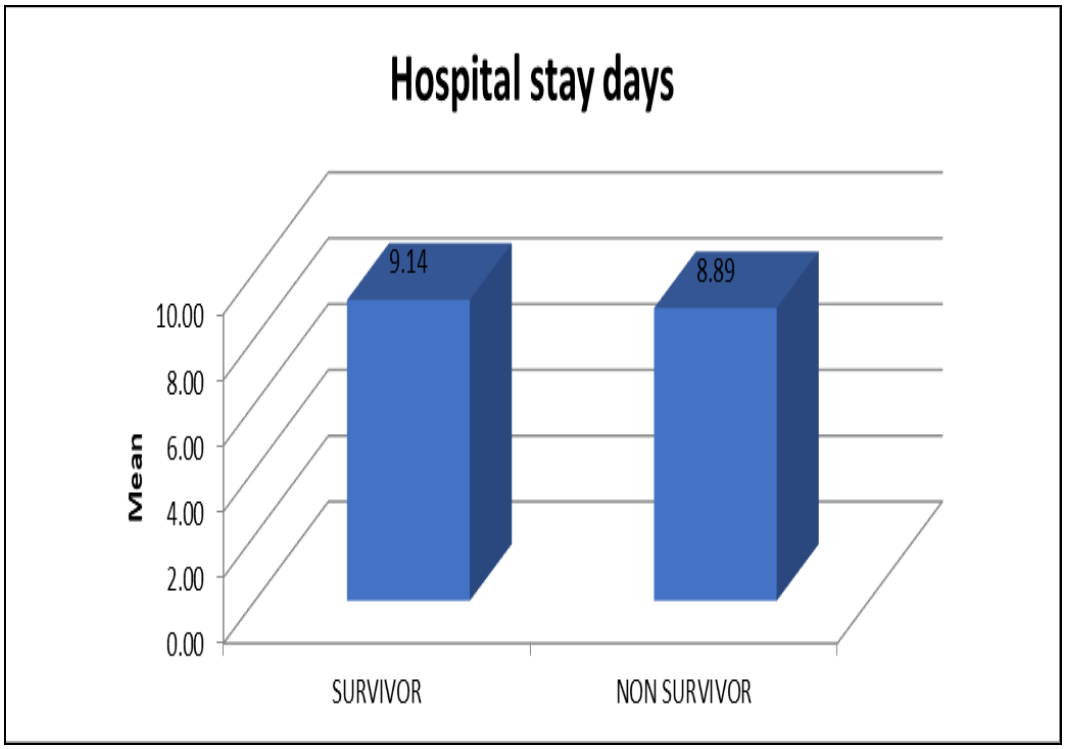


FIGURE 42: COMPARING ICU STAY OF SURVIVOR VS NON-SURVIVOR

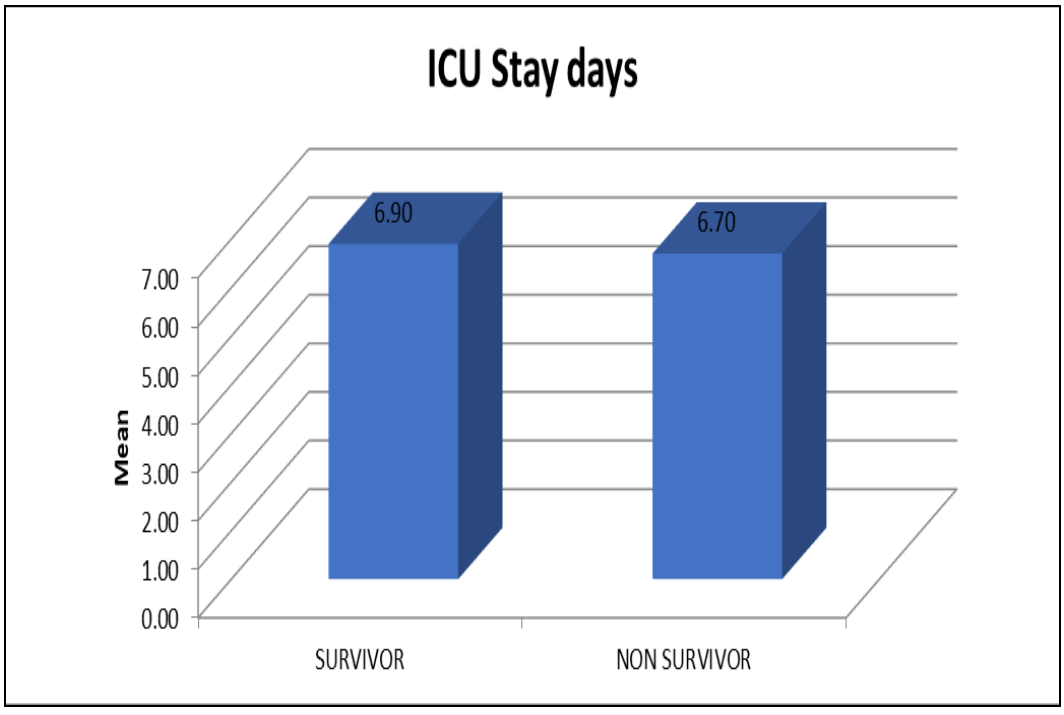


TABLE 40: COMPARISON OF AGE GROUPS BETWEEN SURVIVOR VS NON-SURVIVOR

Age Groups	SURVIVOR		NON-SURVIVOR		p-value
<30	38	12.38%	9	12.33%	0.90
30-39	43	14.01%	10	13.70%	
40-49	100	32.57%	27	36.99%	
50-59	88	28.66%	17	23.29%	
>60	38	12.38%	10	13.70%	

There was no difference between survivor and non- survivor with respect to various age groups. Result was statistically not significant on Chi-Square test. On Kaplan-Meier survival analysis, survival probability was also statistically not significant**.**

FIGURE 43: COMPARISON OF AGE GROUPS BETWEEN SURVIVOR VS NON-SURVIVOR

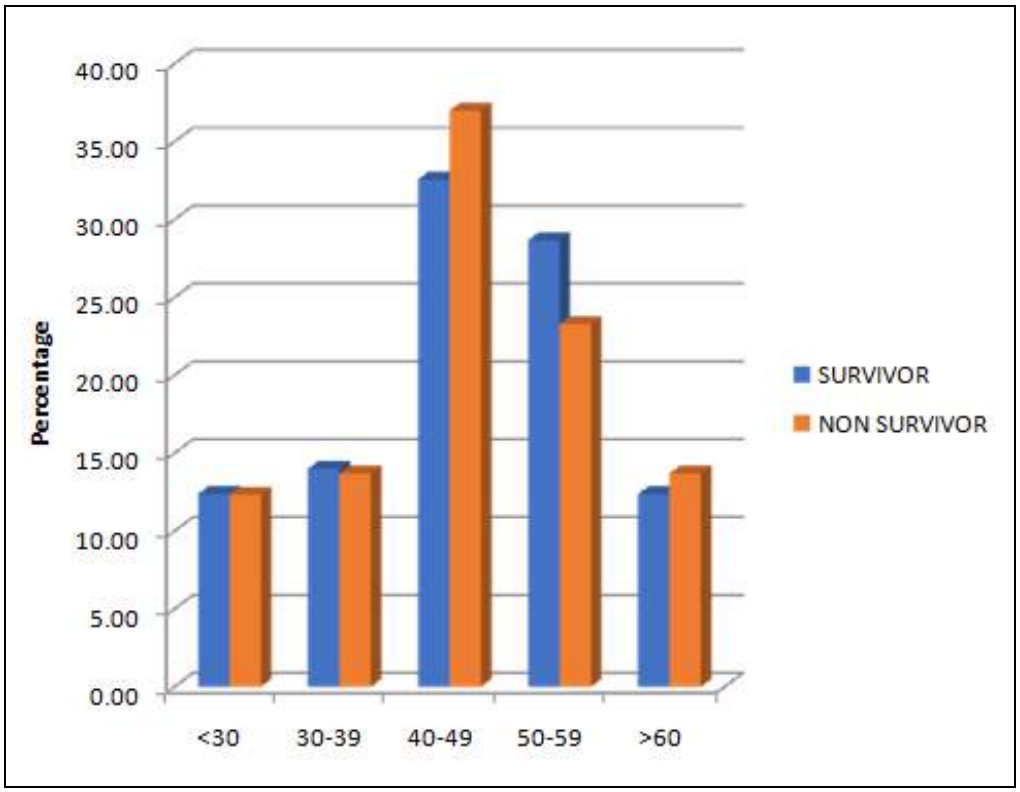


FIGURE 44: SURVIVAL PROBABILITY OF DIFFERENT AGE GROUPS

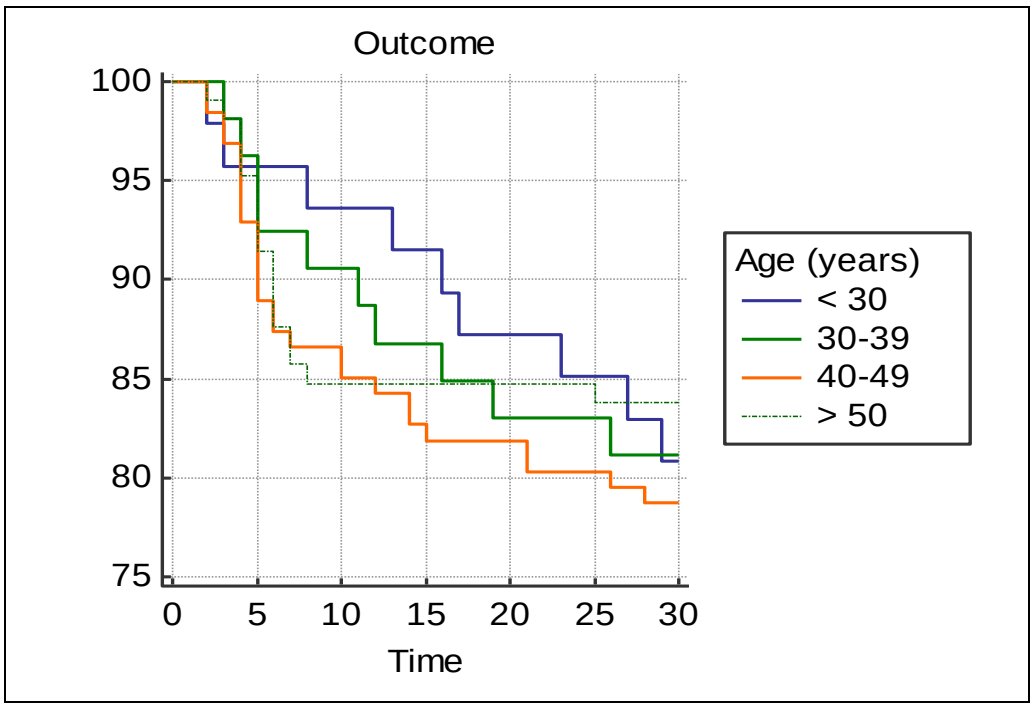


TABLE 41: COMPARING SURVIVORS AND NON-SURVIVORS FOR GENDER

Gender	SURVIVOR		NON-SURVIVOR		p-value
F	128	41.69	30	41.10	0.92
M	179	58.31	43	58.90	

There was no difference between the 2 genders with respect to percentage cases of survivors' vs non-survivors. Also, on survival analysis, both genders had similar survival rate.

FIGURE 45: COMPARING SURVIVORS AND NON-SURVIVORS FOR GENDER

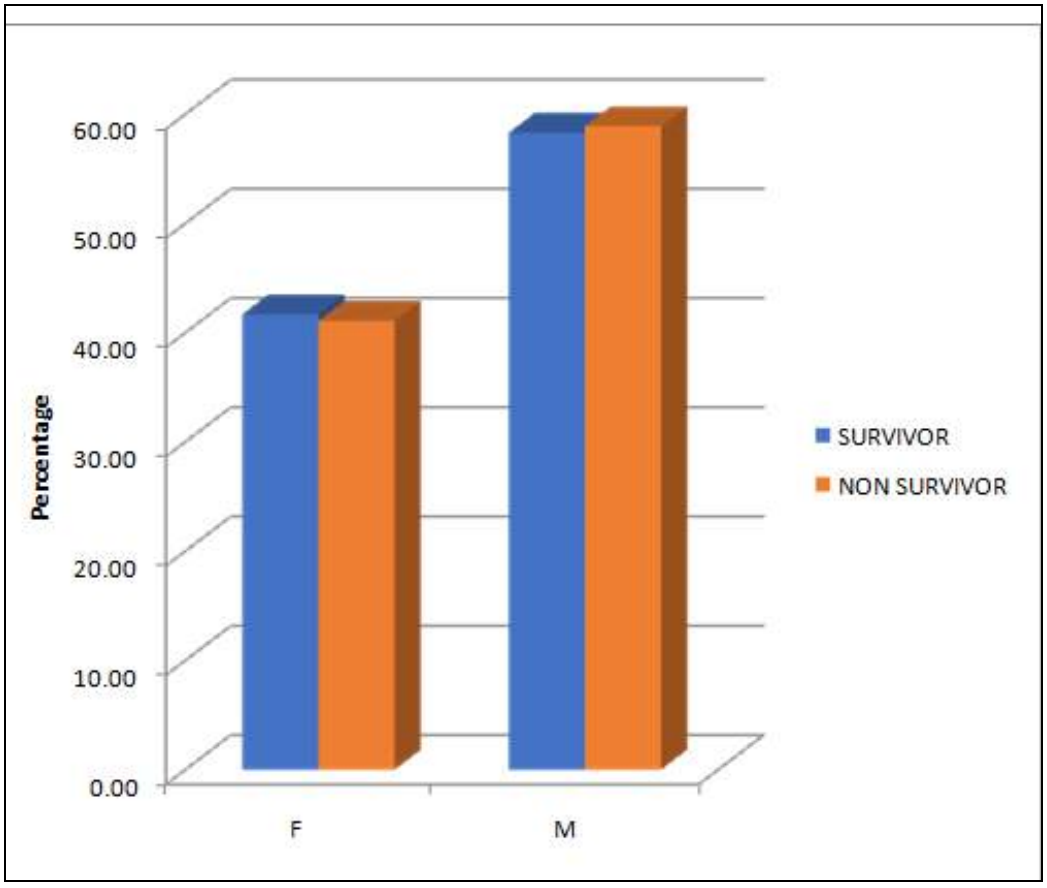


FIGURE 46: SURVIVAL PROBABILITY ACCORDING TO GENDER

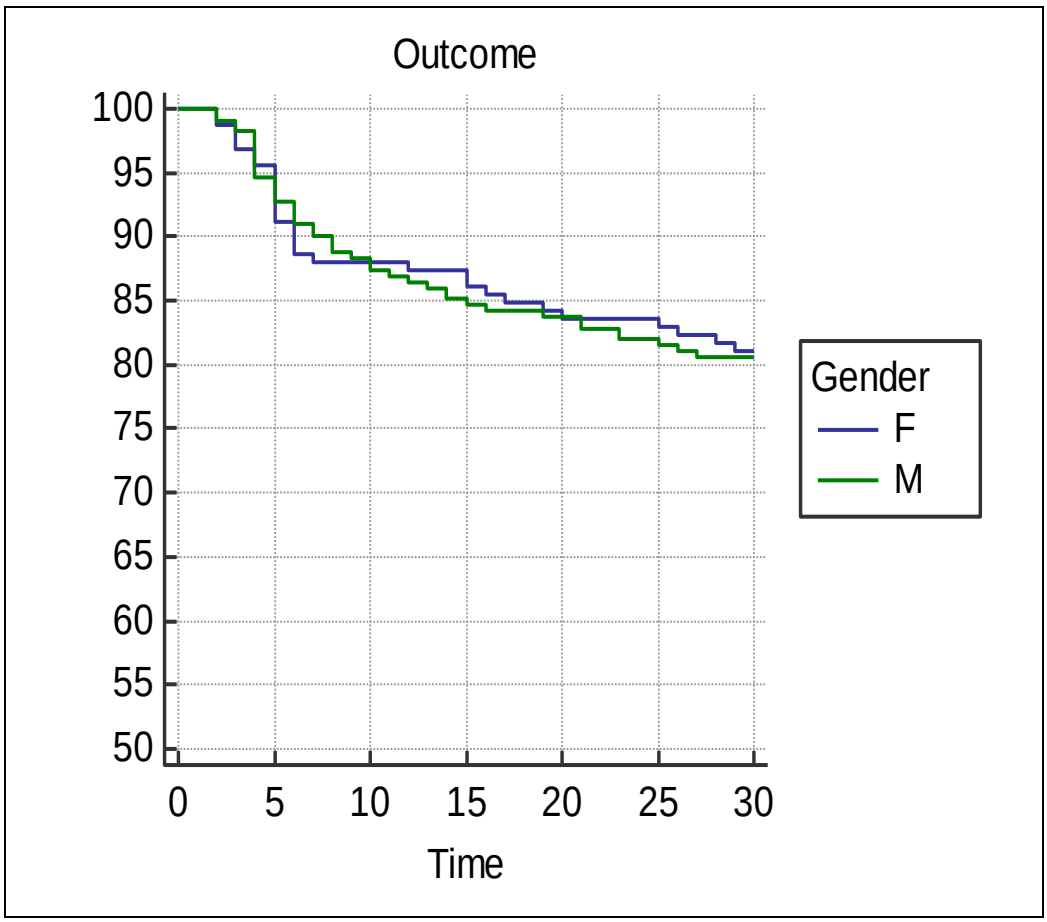


TABLE 42: COMPARING SURVIVORS AND NON-SURVIVORS FOR RESIDENTIAL STATUS

Rural/Urban	SURVIVOR		NON-SURVIVOR		p-value
RURAL	177	57.65	44	60.27	0.68
URBAN	130	42.35	29	39.73	

There was no difference between the rural and urban dwellers with respect to percentage cases of survivors' vs non-survivors. Also, on survival analysis, both groups had similar survival rate.

FIGURE 47: COMPARING SURVIVORS AND NON-SURVIVORS FOR RESIDENTIAL STATUS

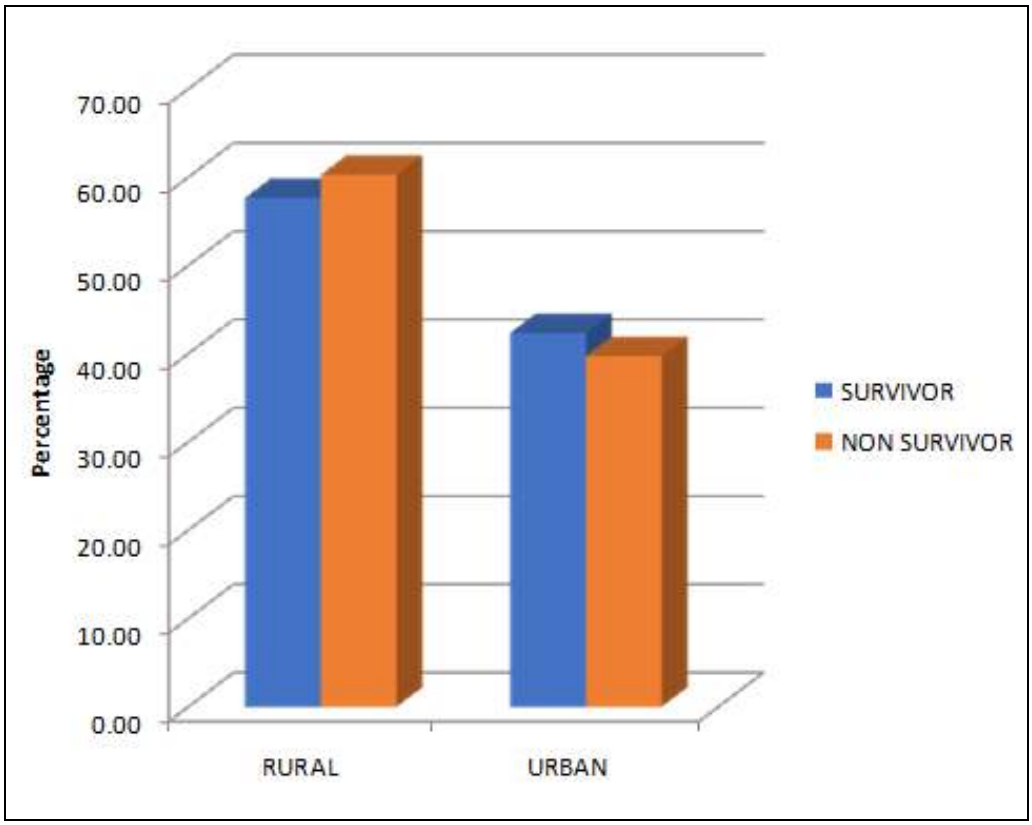


FIGURE 48: SURVIVAL PROBABILITY ACCORDING TO RESIDENTIAL STATUS

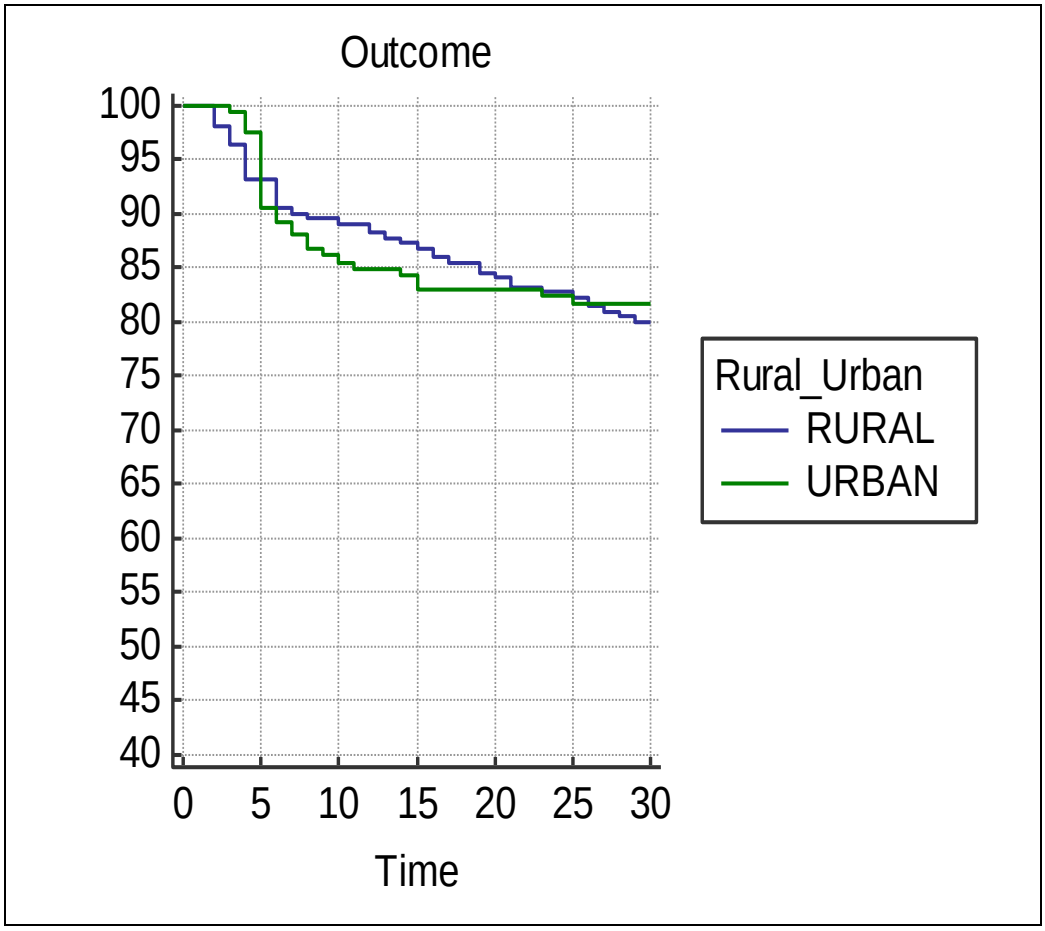


TABLE 43: COMPARING SURVIVORS AND NON-SURVIVORS FOR NUMBER OF COMORBITIDIES

COMORBIDITY GROUP	SURVIVOR		NON-SURVIVOR		p-value
No	138	44.95	25	34.25	0.001
Single	96	31.27	11	15.07	
Two	55	17.92	20	27.40	
≥ Three	18	5.86	17	23.29	

There was statistically significant difference between the various comorbidity groups on chi square test with respect survivors' and non-survivors with a p valve of 0.001. Also, on survival analysis, survival rate was statistically significant between the four groups (p value below 0.05). minimum mortality was in the group with single comorbidity follow by no comorbidity, 2 comorbidity and maximum with group with 3 or more comorbidity.

FIGURE 49: COMPARING SURVIVORS AND NON-SURVIVORS FOR NUMBER OF COMORBITIDIES

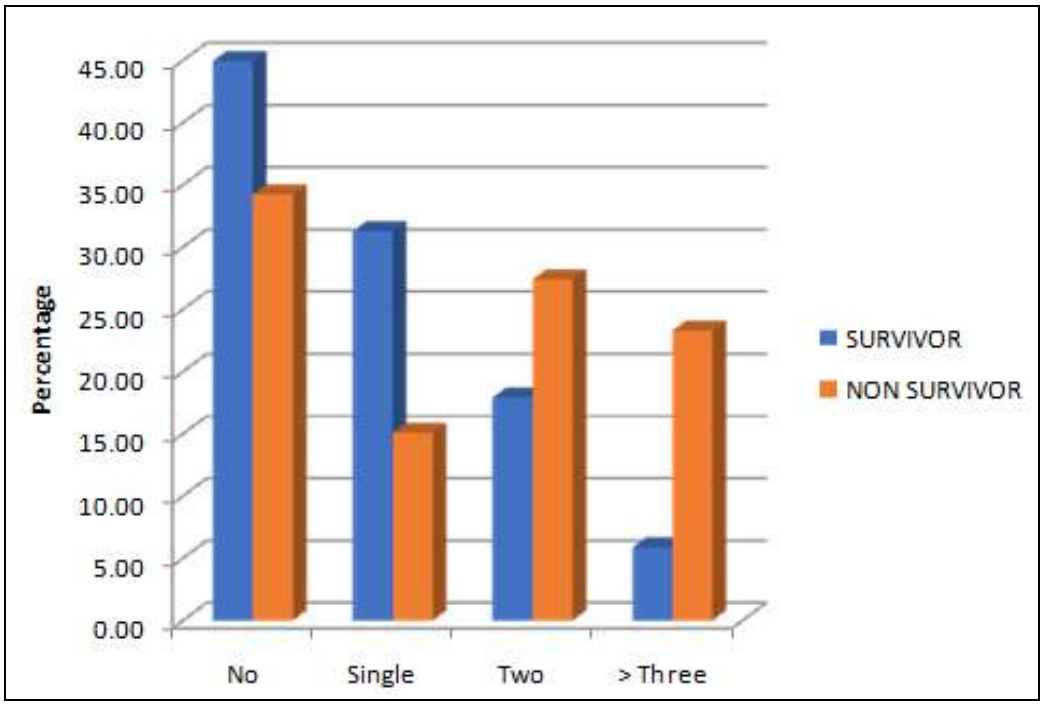


FIGURE 50: SURVIVAL PROBABILITY ACCORDING TO NUMBER OF COMORBIDITIES

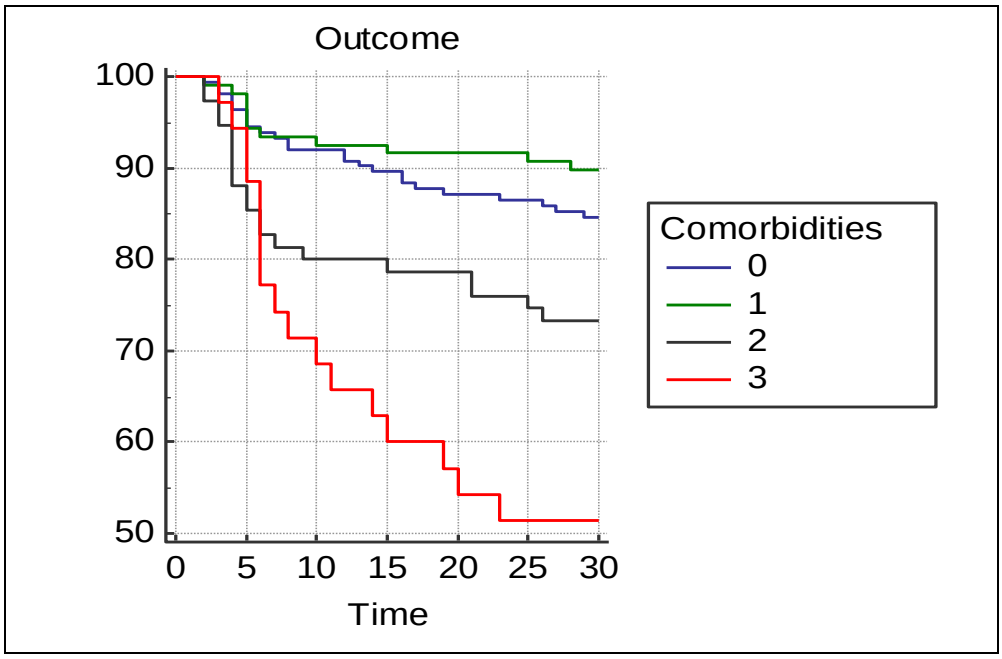


TABLE 44: COMPARING SURVIVORS AND NON-SURVIVORS FOR PATTERN OF AKI

GROUP	SURVIVOR		NON-SURVIVOR		p-value
MED	228	74.27	61	83.56	0.20
OBS	19	6.19	4	5.48	
SUR	60	19.54	8	10.96	

Though there was difference between the survivors and non-survivor's with Medical AKI having higher percentage of mortality, but it was not statistically significant with a p value of 0.20.

survival analysis showed that most of the mortality in obstetric group were early mortality while other groups had both early as well as late mortalities. Still the result didn't reach statistical significance.

FIGURE 51: COMPARING SURVIVORS AND NON-SURVIVORS FOR PATTERN OF AKI

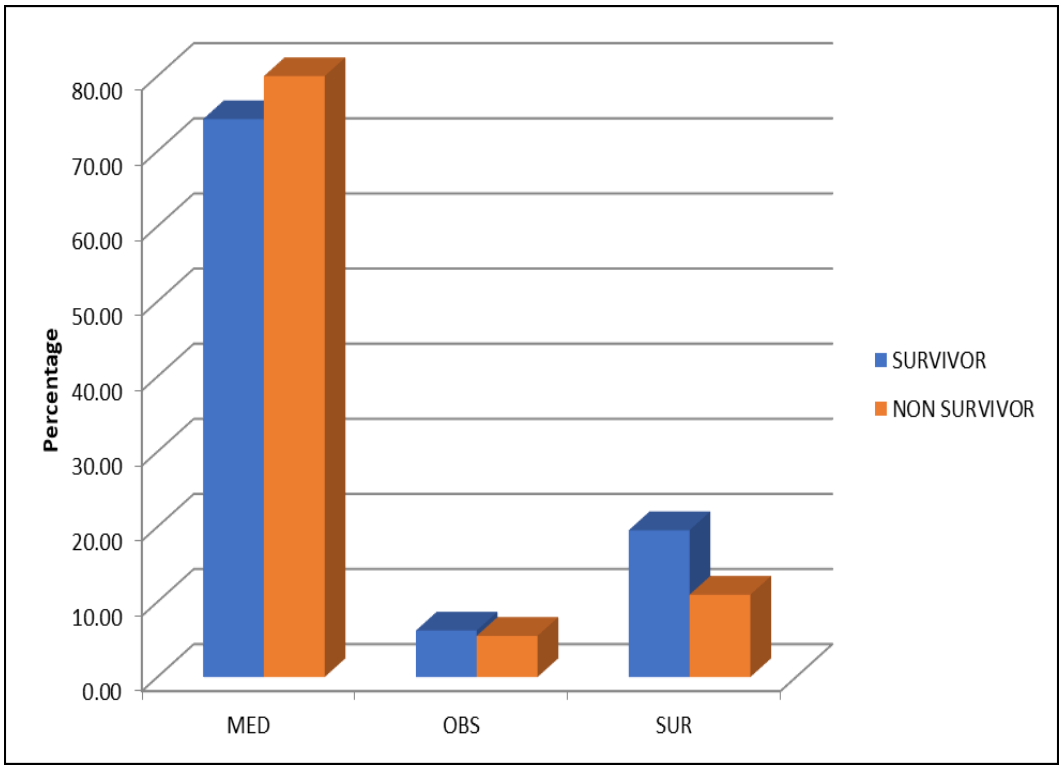


FIGURE 52: SURVIVAL PROBABILITY ACCORDING TO PATTERN OF AKI

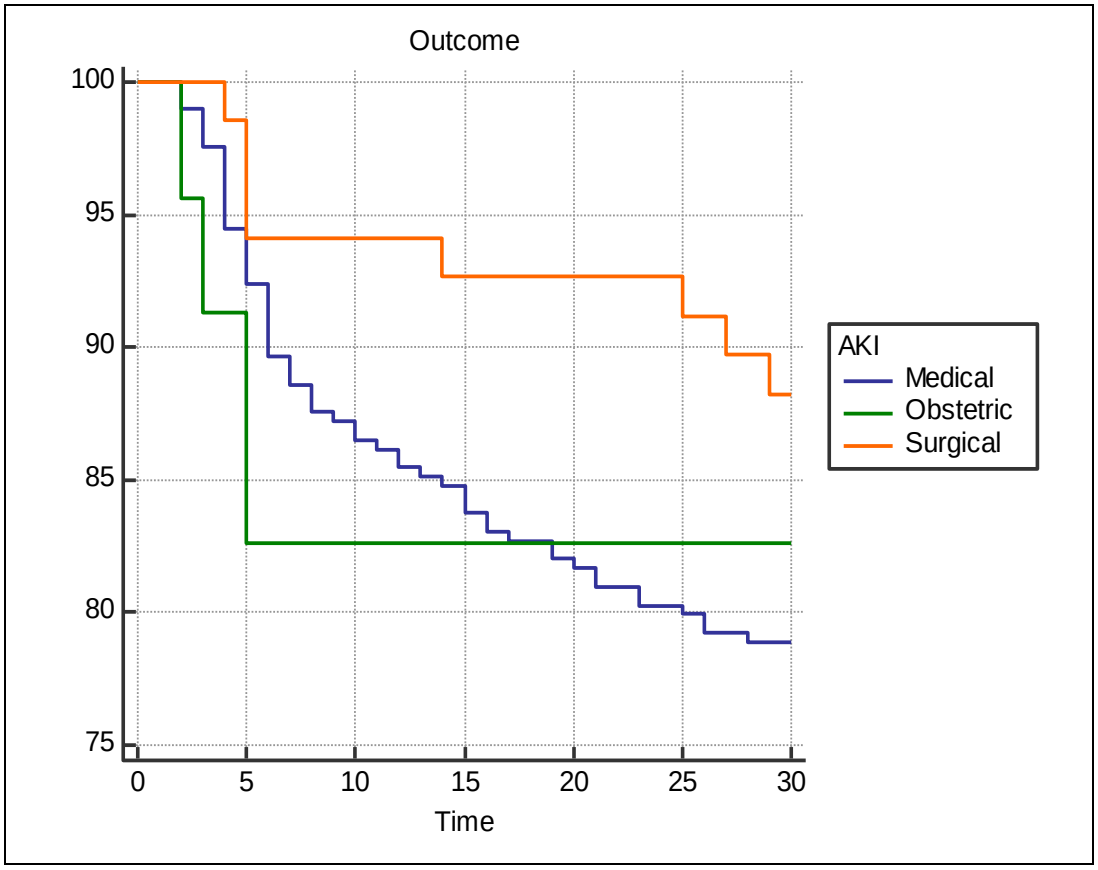


TABLE 45: COMPARING SURVIVORS AND NON-SURVIVORS FOR INFECTIOUS ETIOLOGY

INFECTIVE ETIOLOGY	SURVIVOR		NON-SURVIVOR		p-value
YES	108	35.18	47	64.38	0.01
NO	199	64.82	26	35.62	

Infectious etiologies were associated with higher number of mortalities which was statistically significant on chi square analysis. Patients with CA-AKI due to infectious etiologies had 3 times more chance of dying in comparison to patients with CA-AKI due to non-infectious etiologies. Survival analysis showed widely separated worms of infectious and non-infectious etiologies of CA-AKI with statistically significant difference.

FIGURE 53: COMPARING SURVIVORS AND NON-SURVIVORS FOR INFECTIOUS ETIOLOGY

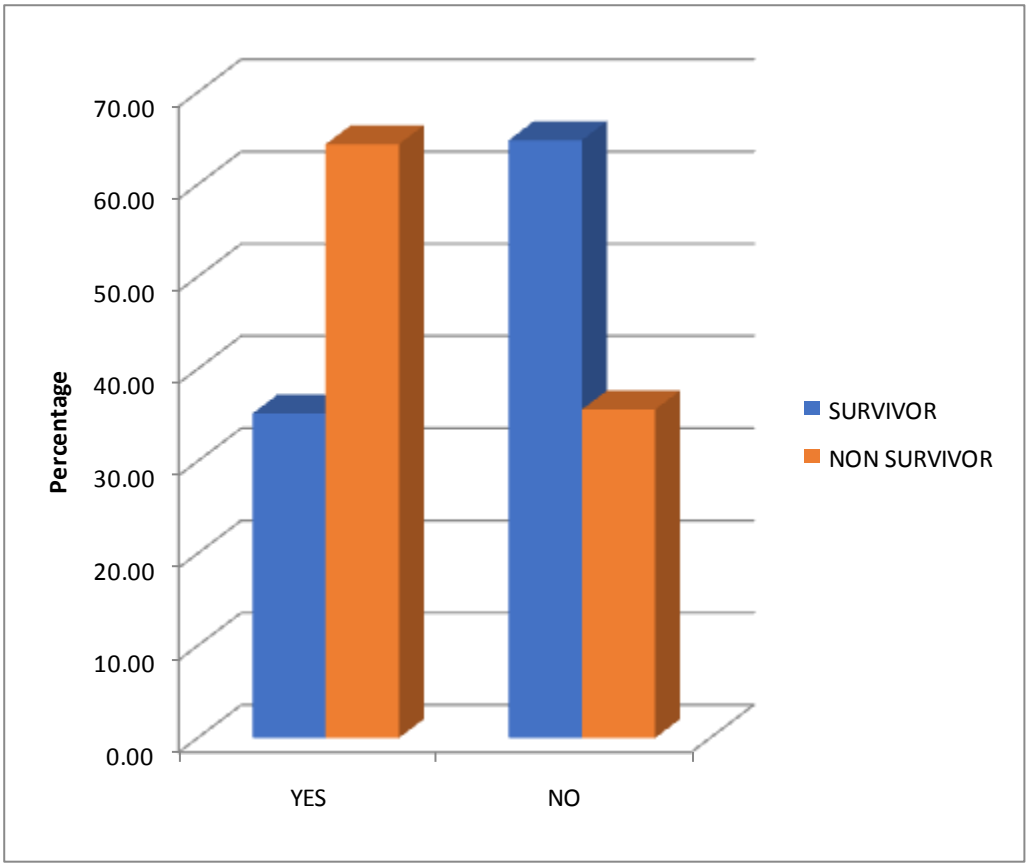


FIGURE 54: SURVIVAL PROBABILITY ACCORDING TO INFECTIOUS ETIOLOGY OF AKI

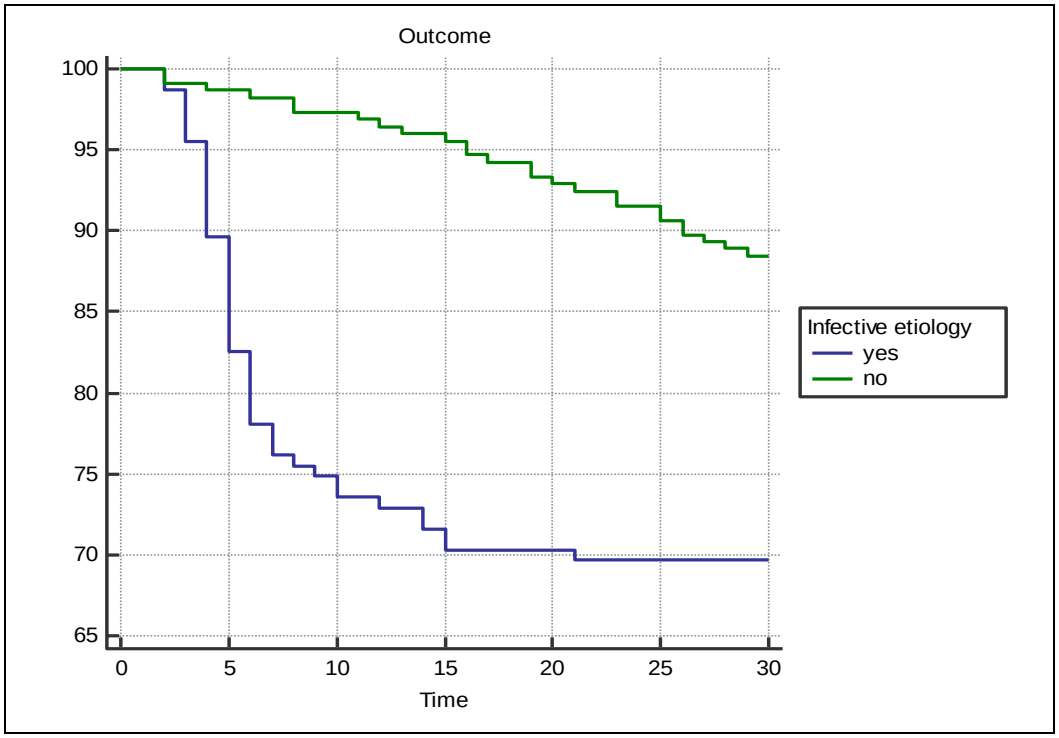


TABLE 46: COMPARING SURVIVORS AND NON-SURVIVORS FOR TROPICAL AND NON-TROPICAL ETIOLOGIES IN AGE BELOW 40 YEARS

TROPICAL ETIOLOGY AGE < 40	SURVIVOR		NON-SURVIVOR		p-value
YES	45	55.56	16	84.21	0.03
NO	36	44.44	3	15.79	

Tropical causes of AKI lead to a greater number of mortalities in age below 40 years where most people were previously healthy (without comorbidity) in comparison to non-tropical causes and this was statistically significant and also well depicted in the survival analysis curve.

FIGURE 55: COMPARING SURVIVORS AND NON-SURVIVORS FOR TROPICAL AND NON-TROPICAL ETIOLOGIES IN AGE BELOW 40 YEARS

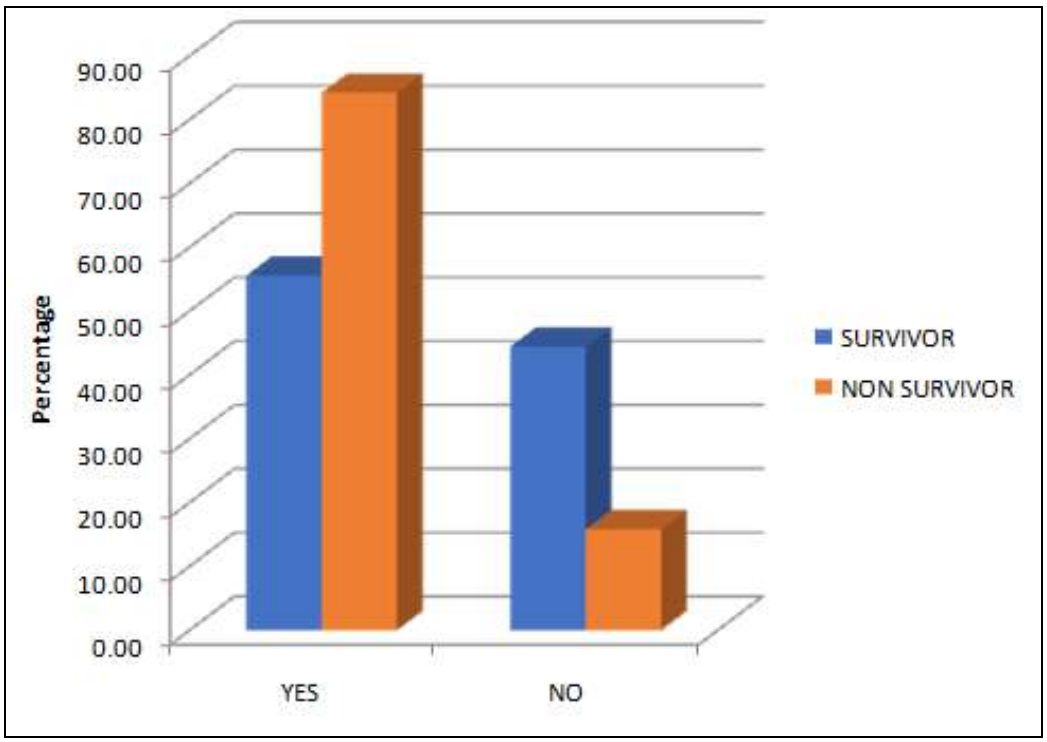


FIGURE 56: SURVIVAL PROBABILITY FOR TROPICAL AND NON-TROPICAL ETIOLOGIES OF AKI IN AGE BELOW 40 YEARS

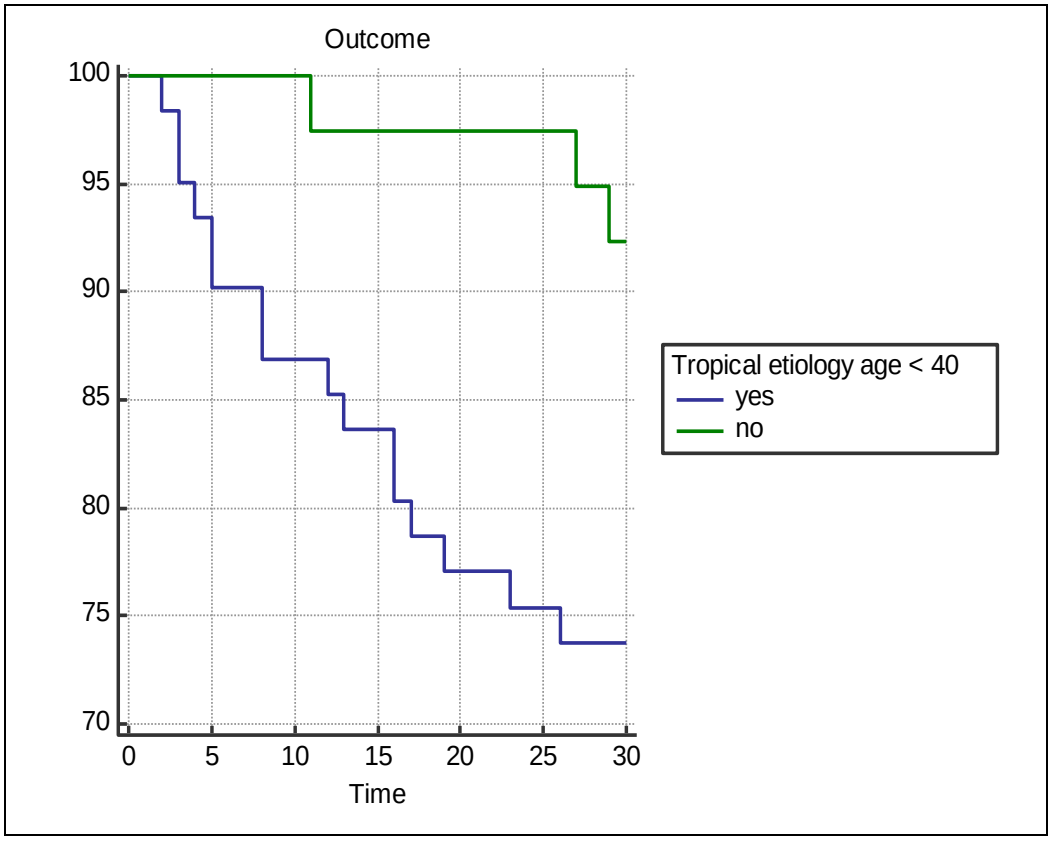


TABLE 47: COMPARING SURVIVORS AND NON-SURVIVORS FOR TROPICAL AND NON-TROPICAL ETIOLOGIES IN AGE ABOVE 40 YEARS

NON- TROPICAL ETIOLOGY AGE > 40	SURVIVOR		NON-SURVIVOR		p-value
YES	103	45.58	35	64.81	0.01
NO	123	54.42	19	35.19	

Opposite to the results for age below 40 years, in age group above 40 years, the mortality was significantly more on the non-tropical etiologies. The results were statistically significant with P value of 0.01. survival analysis also depicted the same.

FIGURE 57: COMPARING SURVIVORS AND NON-SURVIVORS FOR TROPICAL AND NON-TROPICAL ETIOLOGIES IN AGE ABOVE 40 YEARS

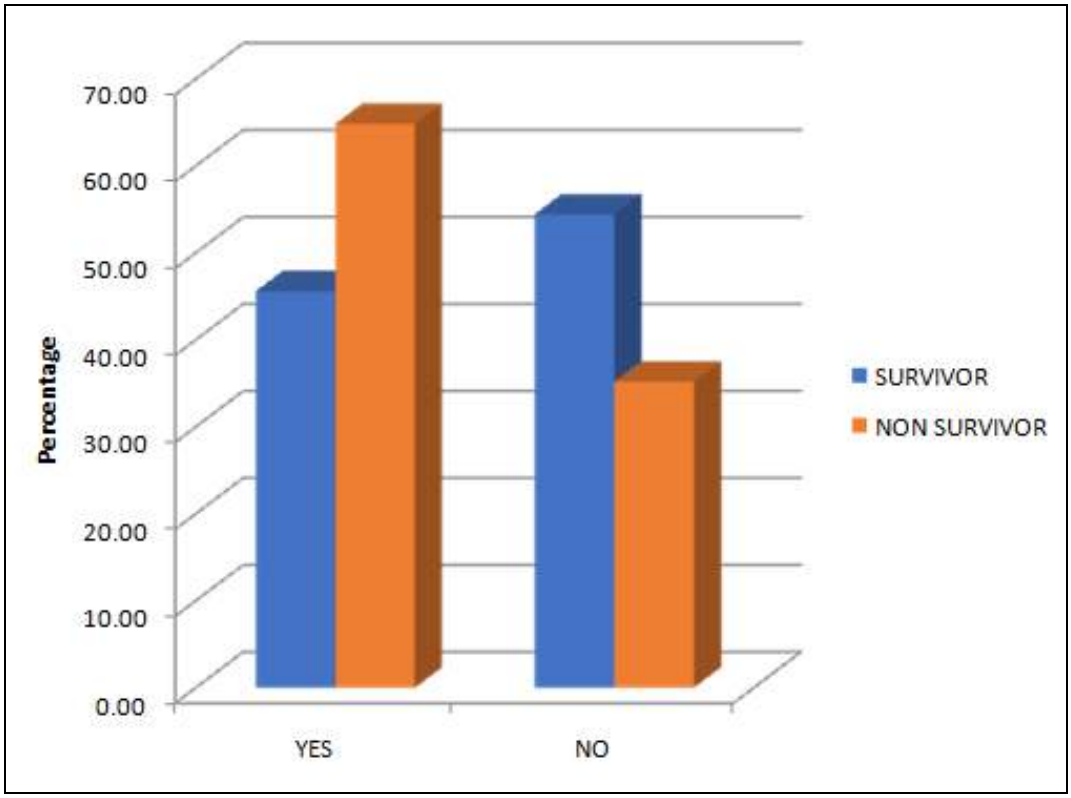


FIGURE 58: SURVIVAL PROBABILITY FOR TROPICAL AND NON-TROPICAL ETIOLOGIES OF AKI IN AGE ABOVE 40 YEARS

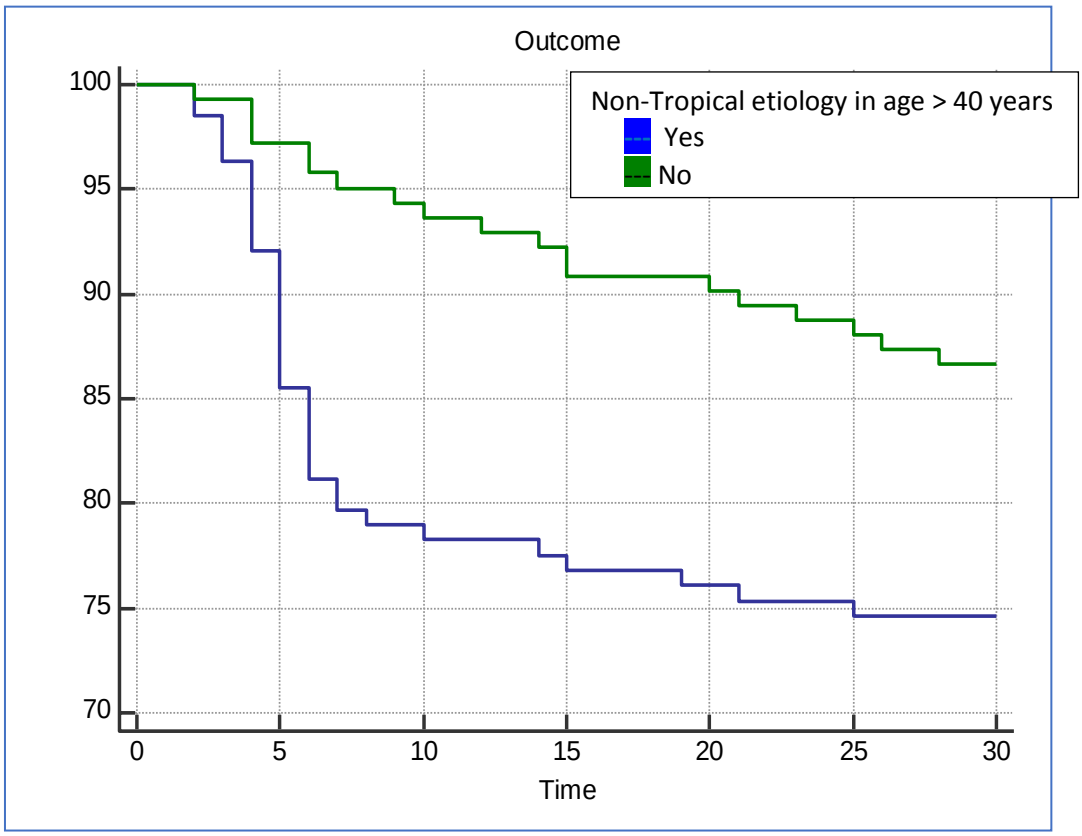


TABLE 48: COMPARING SURVIVORS AND NON-SURVIVORS FOR TYPE OF AKI

TYPE OF AKI	SURVIVOR		NON-SURVIVOR		p-value
HYPOPERFUSION	133	43.32	25	34.25	<0.0001
INTRINSIC	133	43.32	48	65.75	
OBSTRUCTIVE	41	13.36	0	0.00	

There was significantly higher mortality in the intrinsic AKI group in comparison to the other 2 group. Obstructive AKI had no cases of mortality.

FIGURE 59: COMPARING SURVIVORS AND NON-SURVIVORS FOR TYPE OF AKI

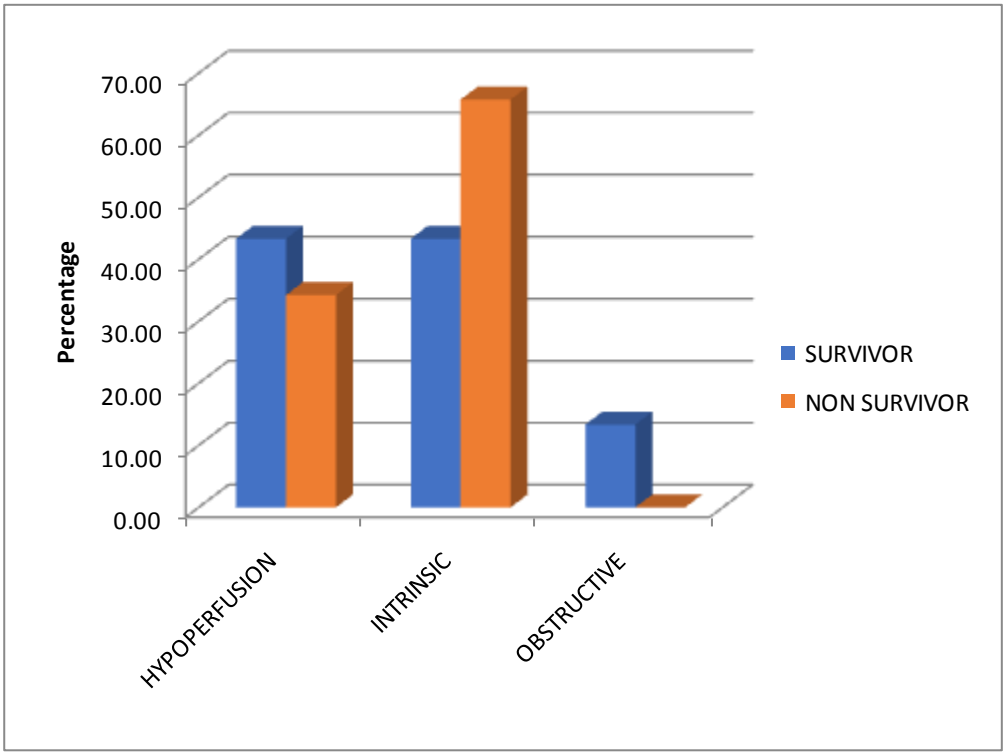


FIGURE 60: SURVIVAL PROBABILITY FOR TYPE OF AKI

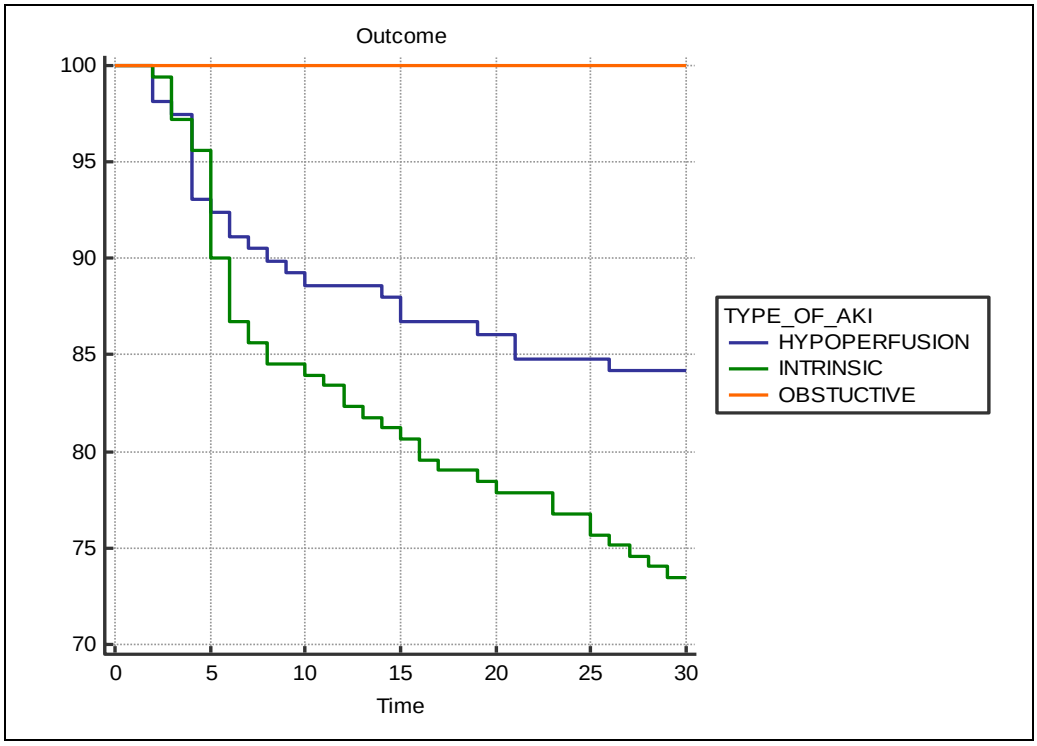


TABLE 49: COMPARING SURVIVORS AND NON-SURVIVORS FOR STAGE OF AKI

Stage	SURVIVOR		NON-SURVIVOR		p-value
1	26	8.5%	0	0.0%	<0.001
2	135	44.0%	4	5.5%	
3	146	47.6%	69	94.5%	

There was significantly higher mortality in the stage 3 AKI group in comparison to the stage 2 and stage 1. The result was significant with p-value below 0.001. There was no mortality in patients with stage 1 AKI.

FIGURE 61: COMPARING SURVIVORS AND NON-SURVIVORS FOR STAGE OF AKI

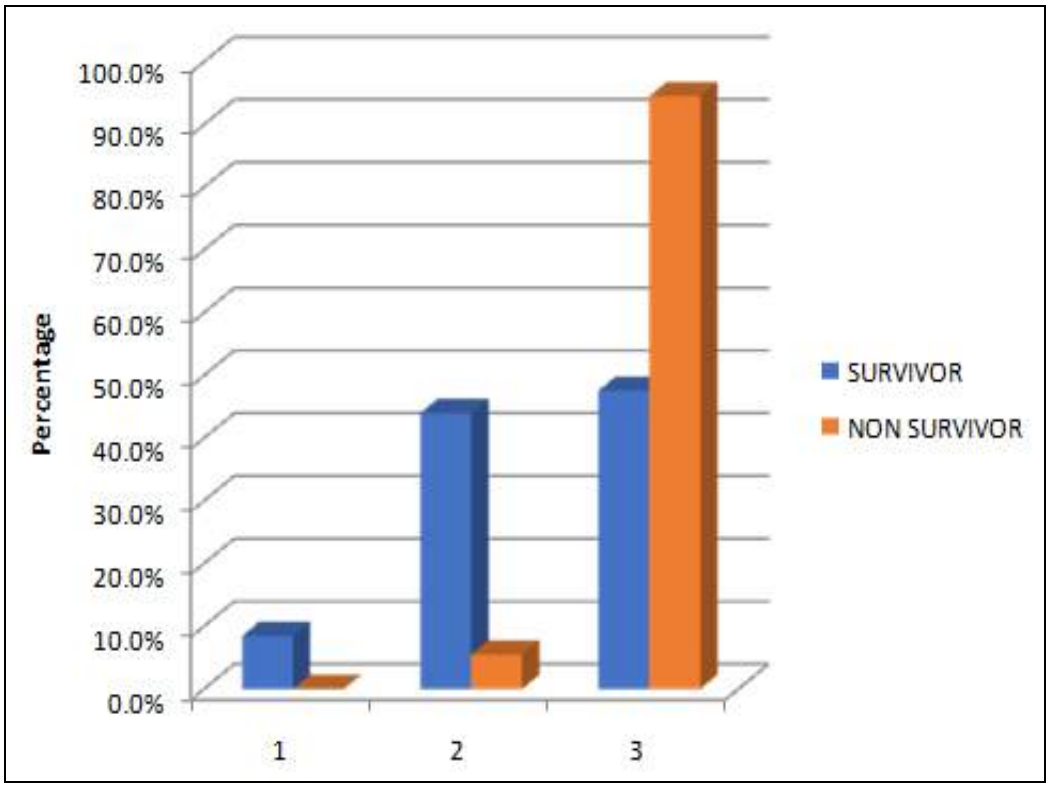


FIGURE 62: SURVIVAL PROBABILITY FOR STAGE OF AKI

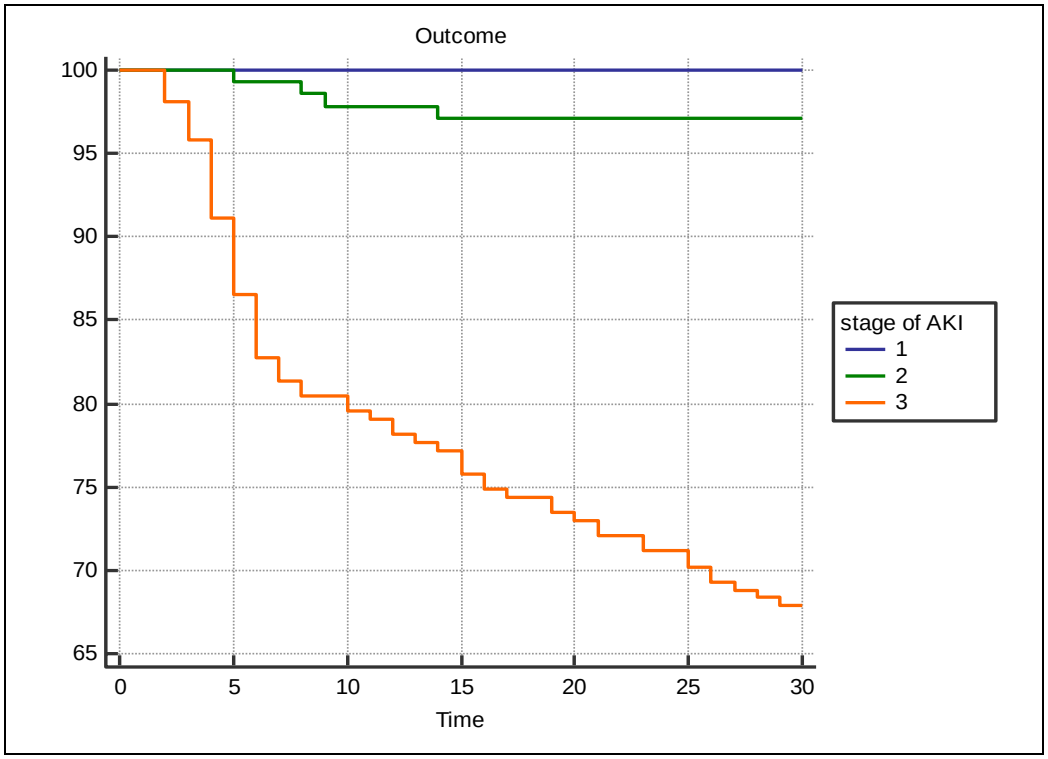


TABLE 50: COMPARING SURVIVORS AND NON-SURVIVORS FOR VARIOUS COMORBIDITIES

COMORBIDITY	SURVIVOR		NON-SURVIVOR		p-value
DM	41	12.70	20	27.40	0.001
HTN	86	28.01	33	45.21	0.004
CKD	70	22.80	36	49.32	<0.00001
CAD	22	7.17	6	8.22	0.75
CLD	17	5.54	13	17.81	<0.001
CVA	12	3.91	7	9.59	0.04
COAD	17	5.54	5	6.85	0.66

In patients developing CA-AKI, diabetics had 33% mortality with 2.1 times more mortality than patient without any comorbidity. It had statistically significant mortality with p value of 0.001. Similarly, hypertensives, patients with underlying CKD, CVA and CLD had statistically more mortality. Patients with CAD and COAD didn't have didn't had significant increase in mortality with CA-AKI in comparison to patients without comorbidity.

FIGURE 63: COMPARING SURVIVORS AND NON-SURVIVORS FOR VARIOUS COMORBIDITIES

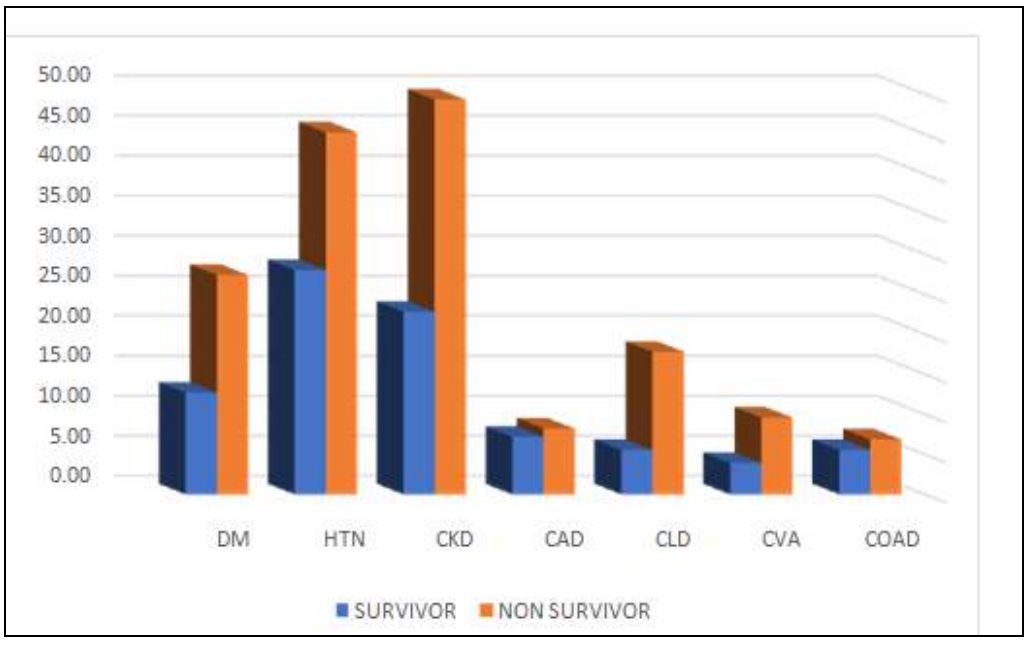


TABLE 51: COMPARING SURVIVORS AND NON-SURVIVORS FOR VARIOUS ETIOLOGIES

ETIOLOGY	SURVIVOR	%	NON SURVIVOR	%	p-value
CARDIORENAL	13	4.23	2	2.74	0.56
CRUSH INJURY	12	3.91	3	4.11	0.93
DRUG	29	9.45	6	8.22	0.74
HYPOVOLUEMIA	71	23.13	2	2.74	<0.0001
POISONING	6	1.95	9	12.33	<0.0001
SEPSIS	66	21.50	34	46.58	<0.0001
SNAKE BITE	6	1.95	2	2.74	0.65
TROPICAL FEVER	42	13.68	13	17.81	0.36
HRS	0	0.00	2	2.74	0.03
U.O.	41	13.36	0	0.00	0.0002
MULTIFACTORIAL AKI	40	13.0	17	23.3	0.027

Etiologies like Sepsis, Poisoning, HRS and multifactorial AKI had significantly higher mortality in comparison to those without these etioogies, with Sepsis and poisoning being highly significant with p-value below 0.001. On the contrary, etiologies like Hypovolemia and obstruction was associated with statistically significant better survival. Other etiologies including cardiorenal, drug induced AKI, crush injury didn't have significant increase in mortality in comparison to total.

On survival analysis, sepsis and poisoning had significant worst outcome. Sepsis and tropical fever were causing more early mortalities while drugs and poisoning especially paraquat caused lot of late mortality.

FIGURE 64: COMPARING SURVIVORS AND NON-SURVIVORS FOR VARIOUS ETIOLOGIES

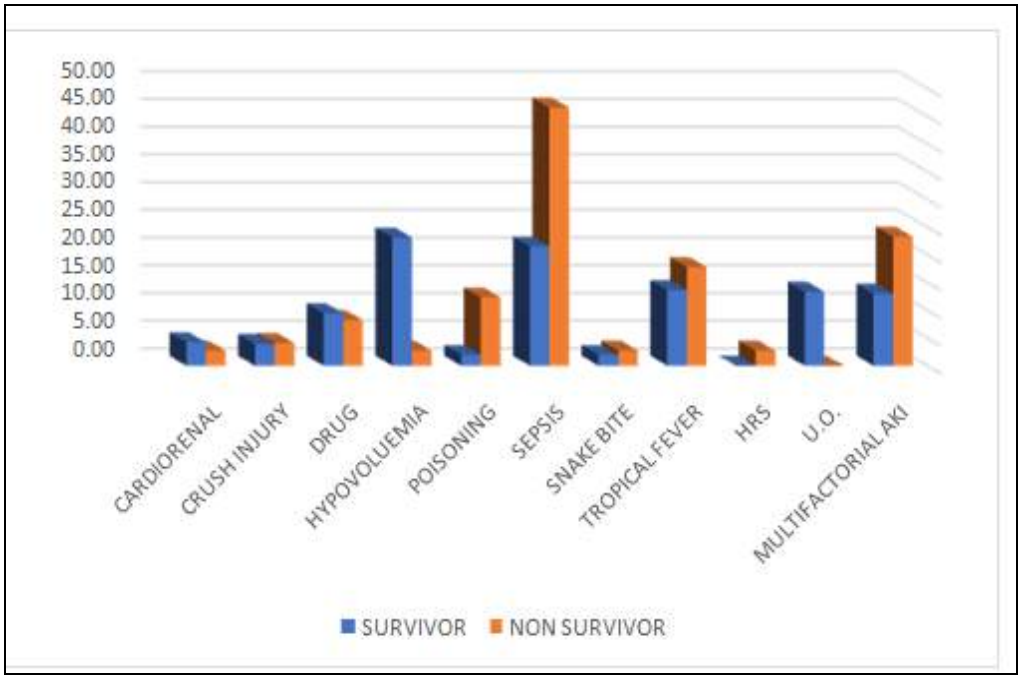


FIGURE 65: SURVIVAL PROBABILITY FOR VARIOUS ETIOLOGIES

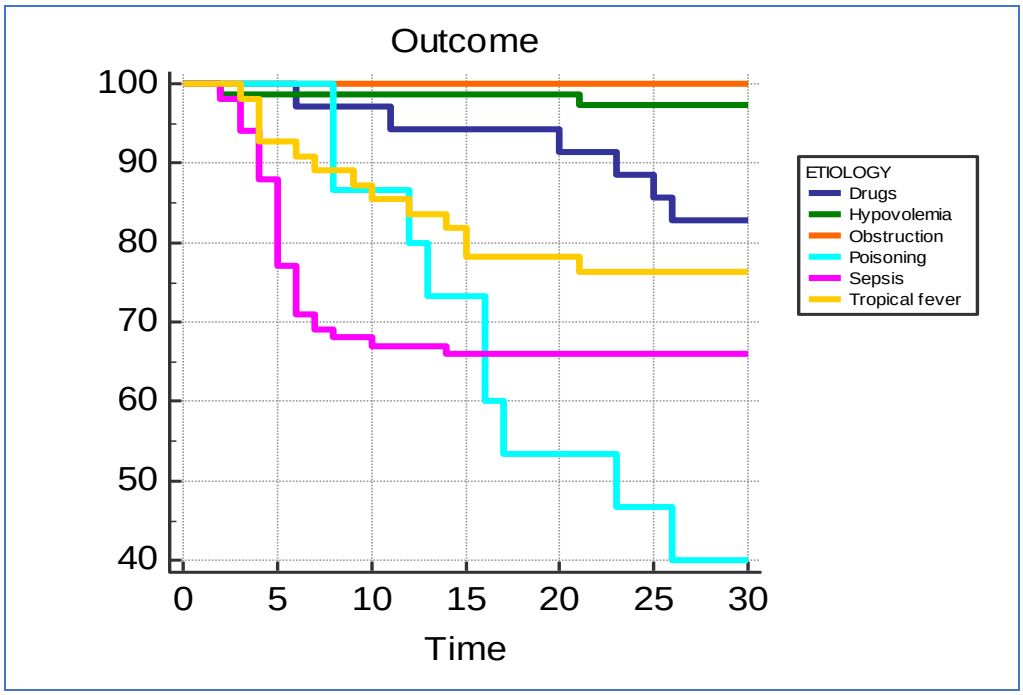


TABLE 52: COMPARING SURVIVORS AND NON-SURVIVORS FOR SEVEARITY OF ILLNESS

	SURVIVOR		NON-SURVIVOR		p-value
NEED OF ICU	91	29.6	68	93.2	0.001
MULTI ORGAN FAILURE	72	23.5	55	75.3	0.01
OLIGURIA	66	21.5	46	63.0	0.001
NEED OF INOTROPES	64	20.8	49	67.1	0.001
NEED OF DIALYSIS	58	18.89	65	89.0	0.001
NEED OF M.VENTILATION	56	18.2	59	80.8	0.01

CA-AKI Patients requiring ICU stay, inotropic support, mechanical ventilation, dialysis, patients who had multiorgan failure, oliguria had significantly poor outcome, higher mortality in comparison to those without it suggesting that severity of underlying comorbidities, etiology and CA-AKI had a significant relation to outcome.

FIGURE 66: COMPARING SURVIVORS AND NON-SURVIVORS FOR SEVEARITY OF ILLNESS

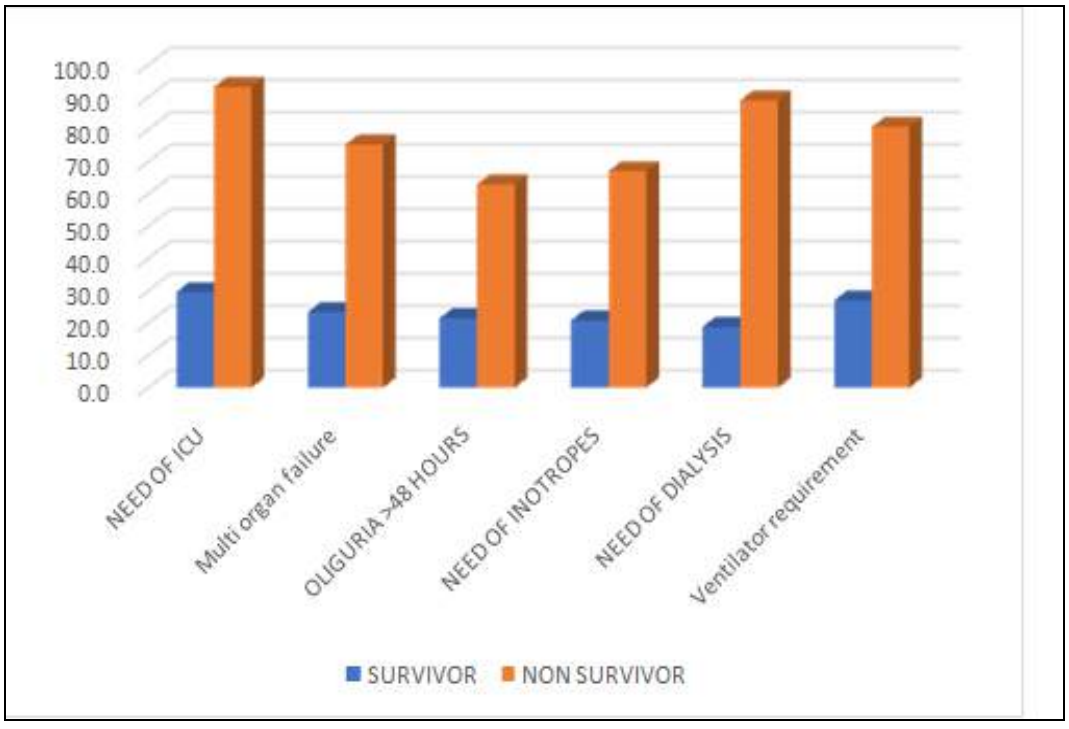


TABLE 53: COMPARING SURVIVORS AND NON-SURVIVORS FOR MODE OF DIALYSIS

MODE OF DIALYSIS	SURVIVOR		NON-SURVIVOR		p-value
CRRT	2	3.4%	6	9.2%	0.001
HD	34	58.6%	16	24.6%	
PD	11	19.0%	17	26.2%	
SLED	11	19.0%	26	40.0%	

Patients requiring CRRT, SLED or PD had significantly higher mortality than those offered HD. There was no statistical difference between outcome of CRRT vs SLED vs PD in our analysis. Survival analysis also showed that the curves of CRRT, SLED and PD are similar with contrast to HD.

FIGURE 67: COMPARING SURVIVORS AND NON-SURVIVORS FOR MODE OF DIALYSIS

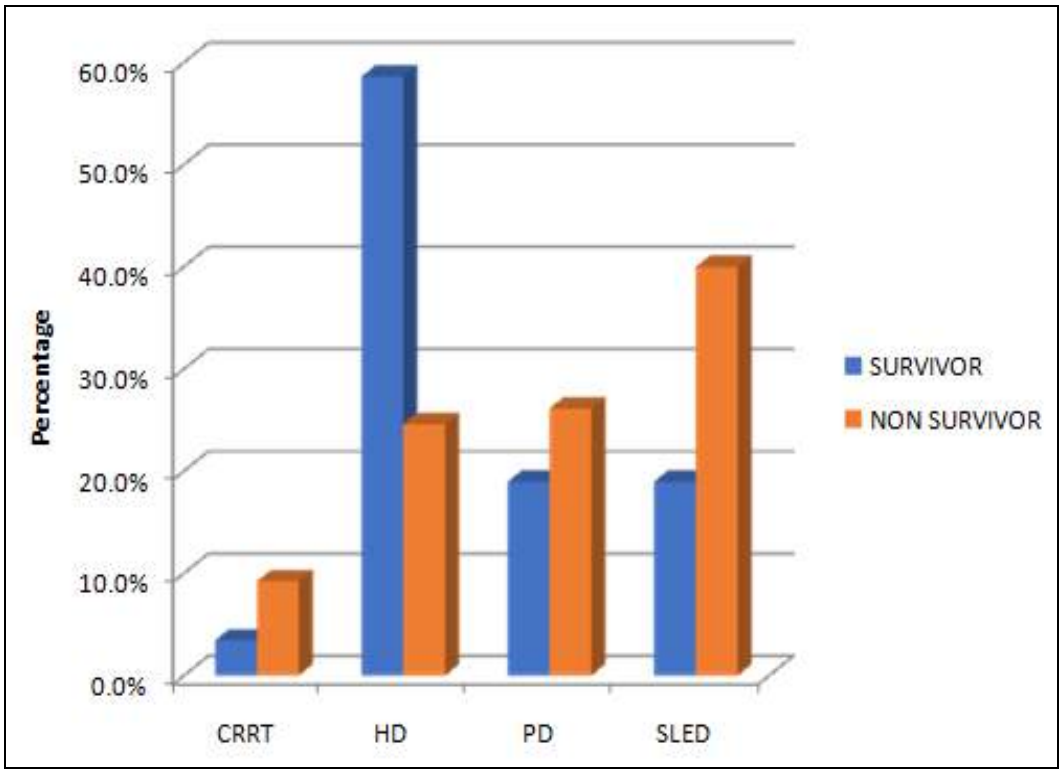
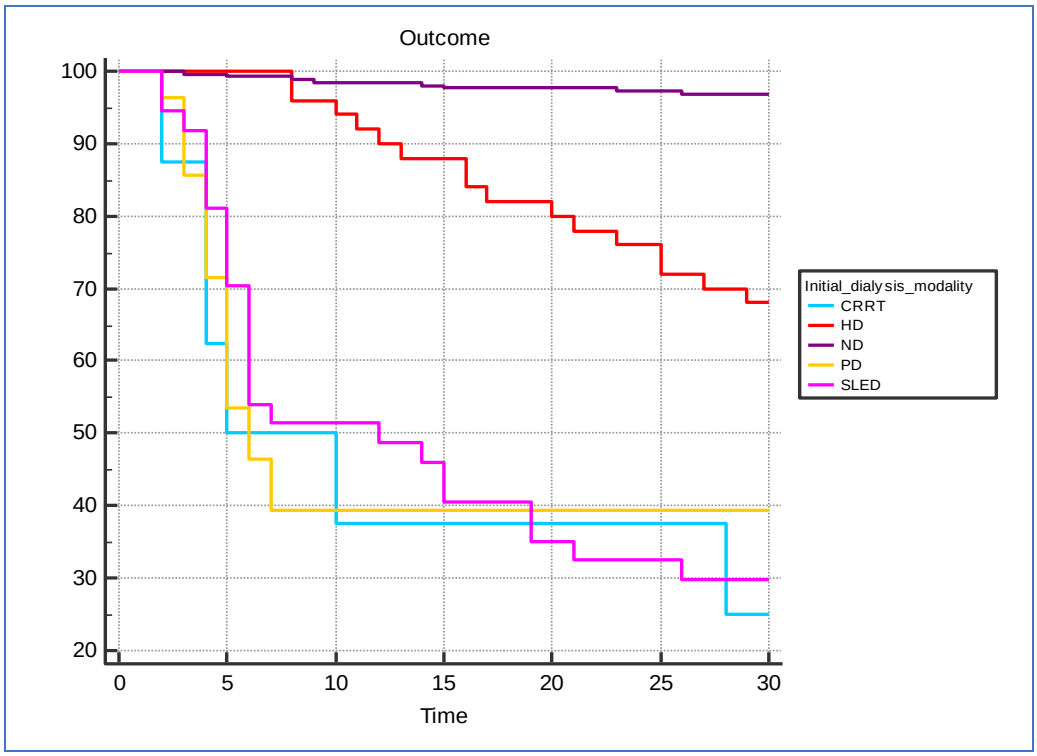


FIGURE 68: SURVIVAL ANALYSIS FOR VARIOUS MODE OF DIALYSIS



FACTORS AFFECTING RECOVERY IN SURVIVORS

TABLE 54: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR AGE, HOSPITAL STAY, AND ICU STAY DURATION

	NR	PR	CR	NR+PR VS CR
CASES	31	103	173	p-value
	NR	PR	CR	
Age years	46.32	49.04	43.13	0.01
Hospital stay days	13.03	10.56	7.6	0.0001
ICU Stay days	6.90	7.23	6.6	0.6

Those who had complete recovery (CR) were younger overall with statistically significant lower mean age in comparison to partial recovery (PR) and non-recovery (NR). CR also had statistically significant shorter hospital stay.

FIGURE 69: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR AGE

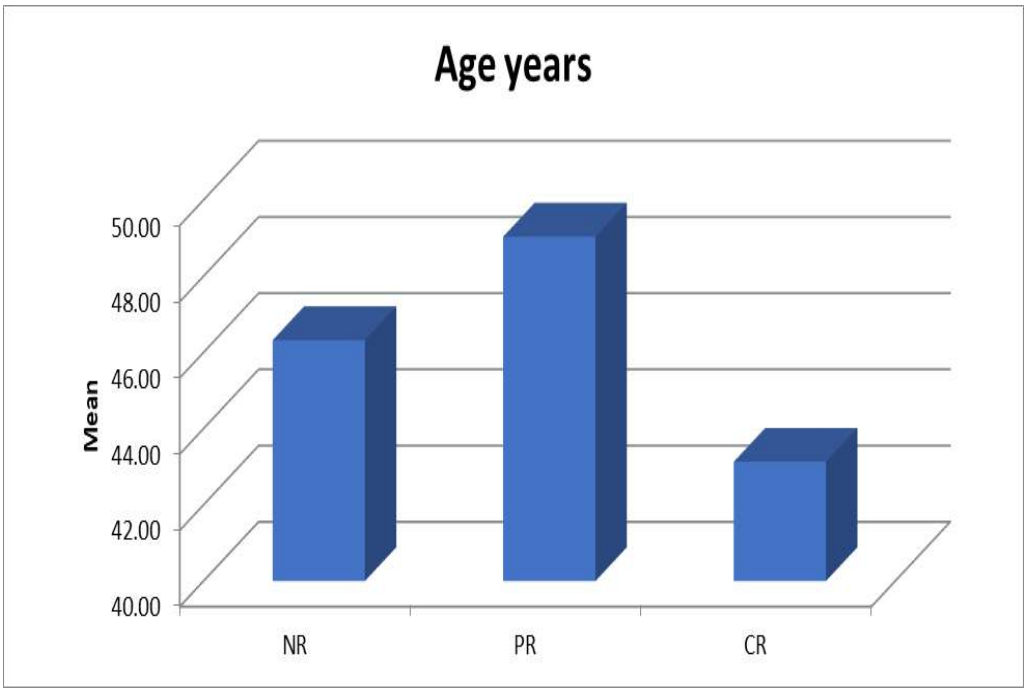


FIGURE 70: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR DURATION OF HOSTIPAL STAY

FIGURE 71: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR DURATION OF ICU STAY

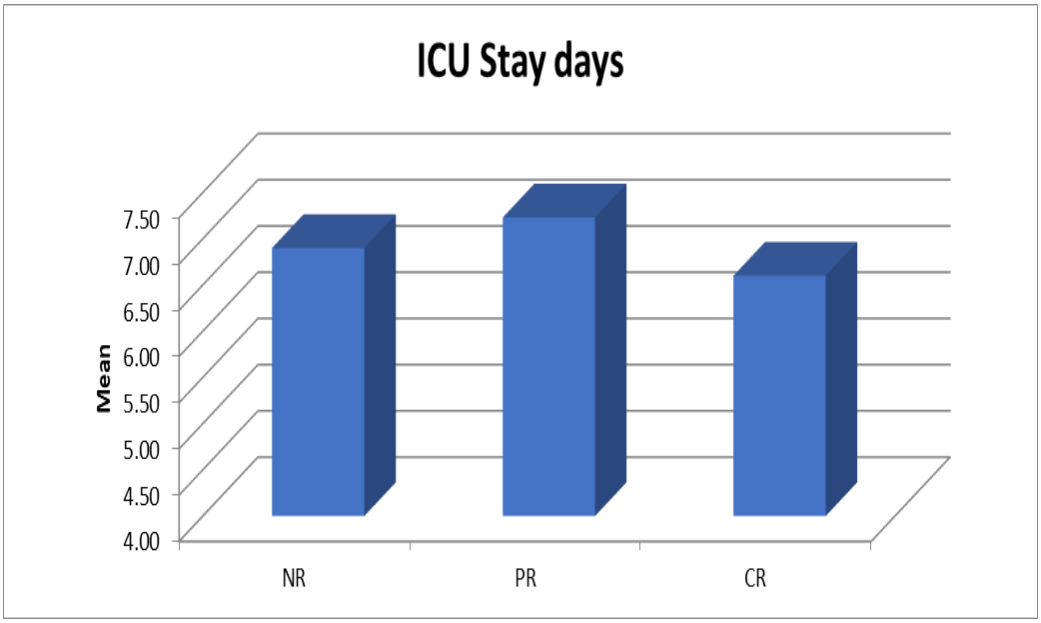


TABLE 55: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR VARIOUS AGE GROUPS

	NR		PR		CR		NR+PR VS CR
Age Groups	No.	%	No.	%	No.	%	p-value
<30	5	16.13	9	8.74	24	13.87	0.004
30-39	2	6.45	8	7.77	33	19.08	
40-49	8	25.81	31	30.10	61	35.26	
50-59	13	41.94	32	31.07	43	24.86	
>60	3	9.68	23	22.33	12	6.94	

Younger the age group, more is the chance of complete recovery. It had statistical significance with a p-value of 0.004

FIGURE 72: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR VARIOUS AGE GROUPS

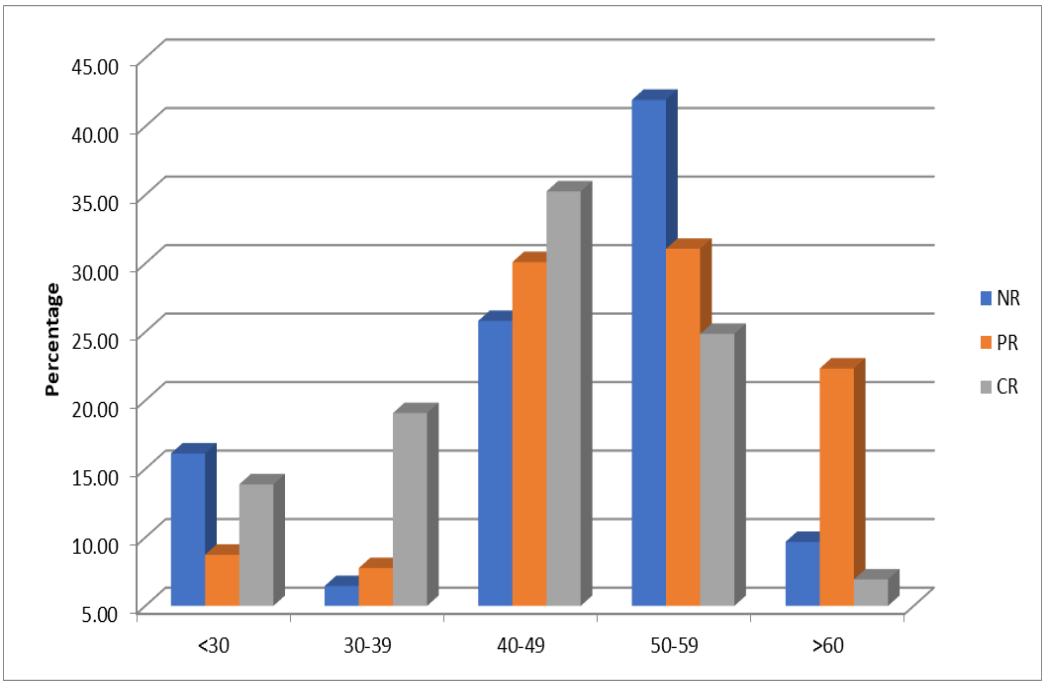


TABLE 56: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR GENDER

	NR		PR		CR		NR+PR VS CR
Gender	No.	%	No.	%	No.	%	p-value
F	14	45.16	37	35.92	77	44.51	0.25
M	17	54.84	66	64.08	96	55.49	

Gender didn't have any significant effect on renal recovery.

FIGURE 73: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR GENDER

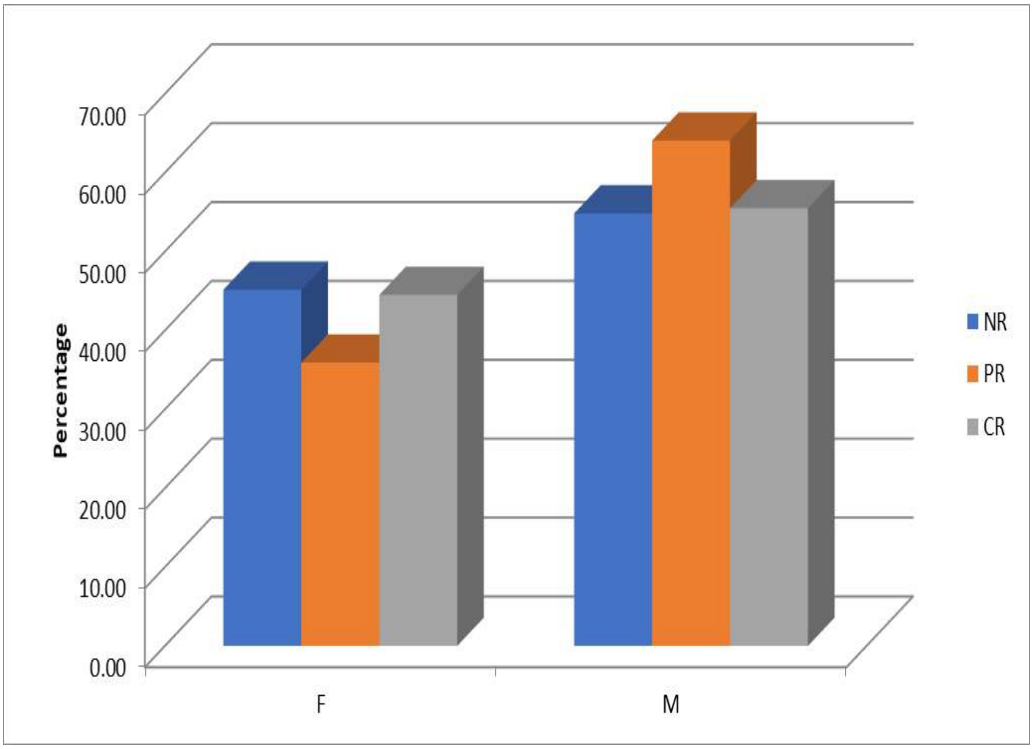


TABLE 57: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR RESIDENTIAL STATUS

	NR		PR		CR		NR+PR VS CR
Rural/Urban	No.	%	No.	%	No.	%	p-value
RURAL	22	70.97	58	56.31	97	56.07	0.52
URBAN	9	29.03	45	43.69	76	43.93	

Though there were higher number of NR in people from rural area, it wasn't statistically significant. Overall residential status didn't affect renal recovery.

FIGURE 74: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR RESIDENTIAL STATUS

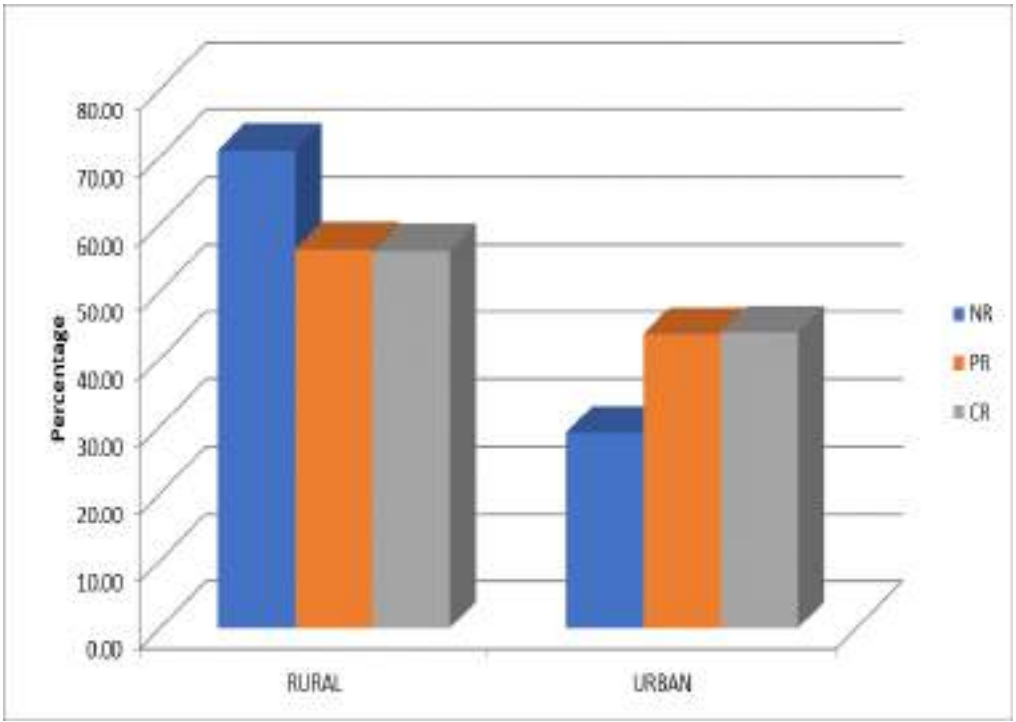


TABLE 58: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR NUMBER OF COMORBIDITIES

		NR		PR		CR	NR+PR VS CR
COMORBIDITY GROUP	No.	%	No.	%	No.	%	p-value
No	8	25.81	26	25.24	104	60.12	<0.0001
Single	9	29.03	35	33.98	52	30.06	
Two	10	32.26	31	30.10	14	8.09	
≥ Three	4	12.90	11	10.68	3	1.73	

There was a very significant relation of renal recovery to lesser number of comorbidities with a p value below 0.001. With increasing number of comorbidities, renal recovery decreases.

FIGURE 75: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR NUMBER OF COMORBIDITIES

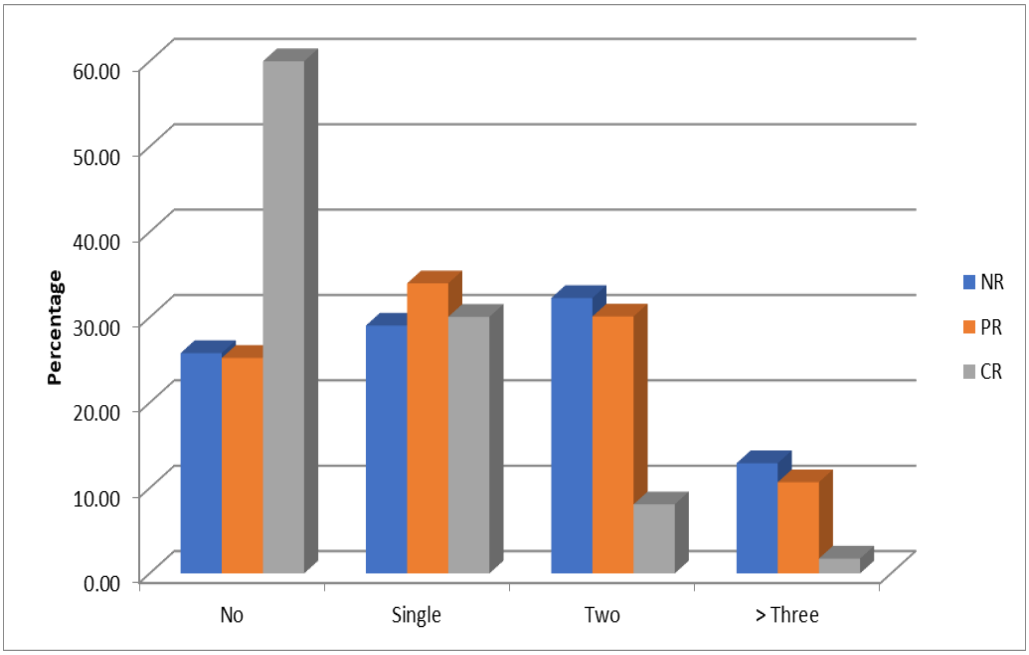


TABLE 59: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR VARIOUS COMORBIDITIES

	NR		PR		CR		NR+PR VS CR
Comorbidity	No.	%	No.	%	No.	%	p-value
DM	9	29.03	24	23.30	6	3.47	<0.0001
HTN	14	45.16	30	29.13	42	24.28	0.09
CKD	10	32.26	49	47.57	11	6.36	<0.0001
CAD	3	9.68	15	14.56	4	2.31	0.0001
CLD	1	3.23	9	8.74	7	4.05	0.1
CVA	3	9.68	4	3.88	5	2.89	0.1
COAD	4	12.90	10	9.71	3	1.73	0.01
NONE	8.00	25.81	26	25.24	104	60.12	0.01

Patients with Diabetes, underlying CKD, CAD and COAD had less chance of complete recovery from the current CA-AKI episode. The statistics were highly significant for underlying Diabetes and CKD. Patient without comorbidities had significantly better chance of recovery

FIGURE 76: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR VARIOUS COMORBIDITIES

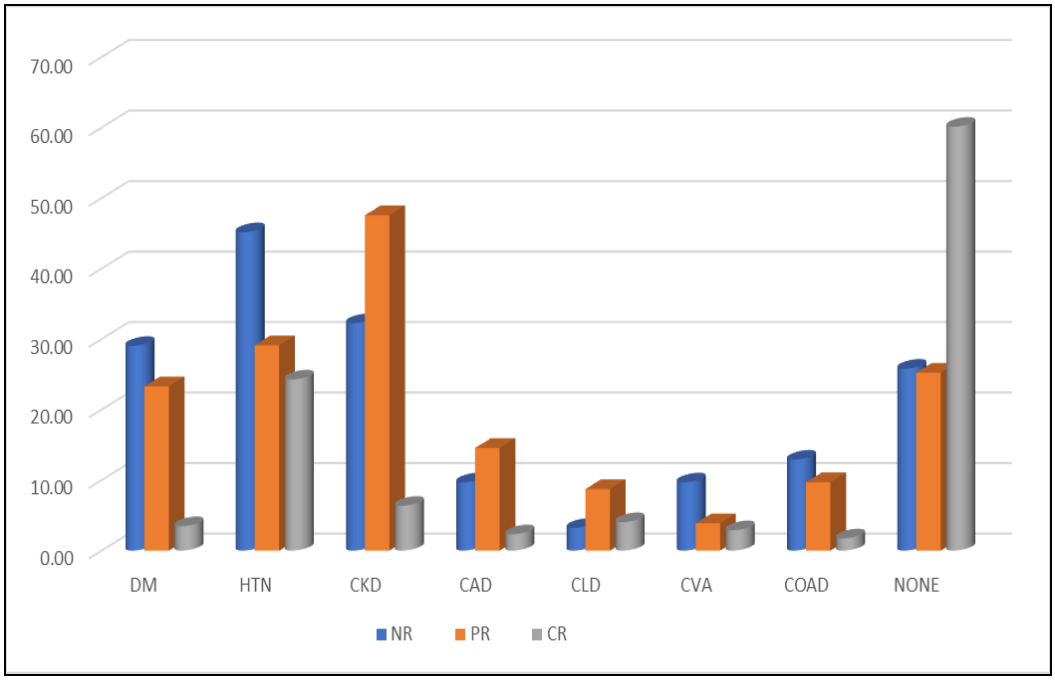


TABLE 60: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR PATTERN OF AKI

	NR		PR		CR		NR+PR VS CR
PRIMARY ETIOLOGY GROUP	No.	%	No.	%	No.	%	p-value
MED	27	87.10	72	69.90	129	74.57	0.12
OBS	3	9.68	2	1.94	14	8.09	
SUR	1	3.23	29	28.16	30	17.34	

Although Medical-AKI had more chance of NR or PR but it was statistically not significant. Overall recovery didn't depend upon medical, surgical or obstetric cause of AKI.

FIGURE 77: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR PATTERN OF AKI

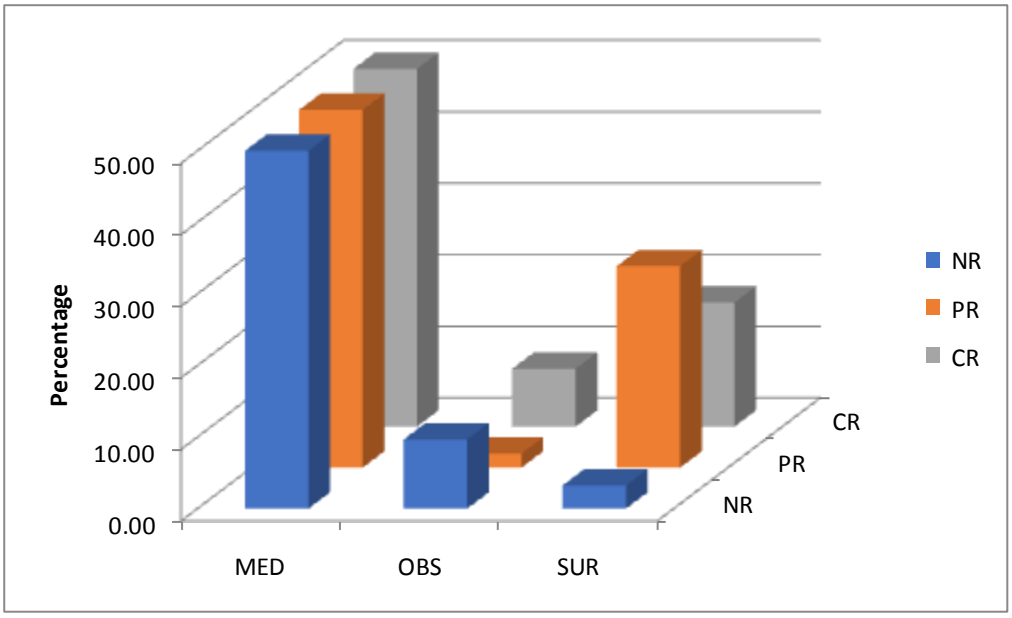


TABLE 61: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR INFECTIVE ETIOLOGY OF AKI

	NR		PR		CR		NR+PR VS CR
INFECTIVE ETIOLOGY	No.	%	No.	%	No.	%	p-value
YES	10	32.26	41	39.81	57	32.95	0.3
NO	21	67.74	62	60.19	116	67.05	

There was no statistically significant difference in renal recovery for infectious vs non-infectious causes of AKI with a P value of 0.3.

FIGURE 78: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR INFECTIVE ETIOLOGY OF AKI

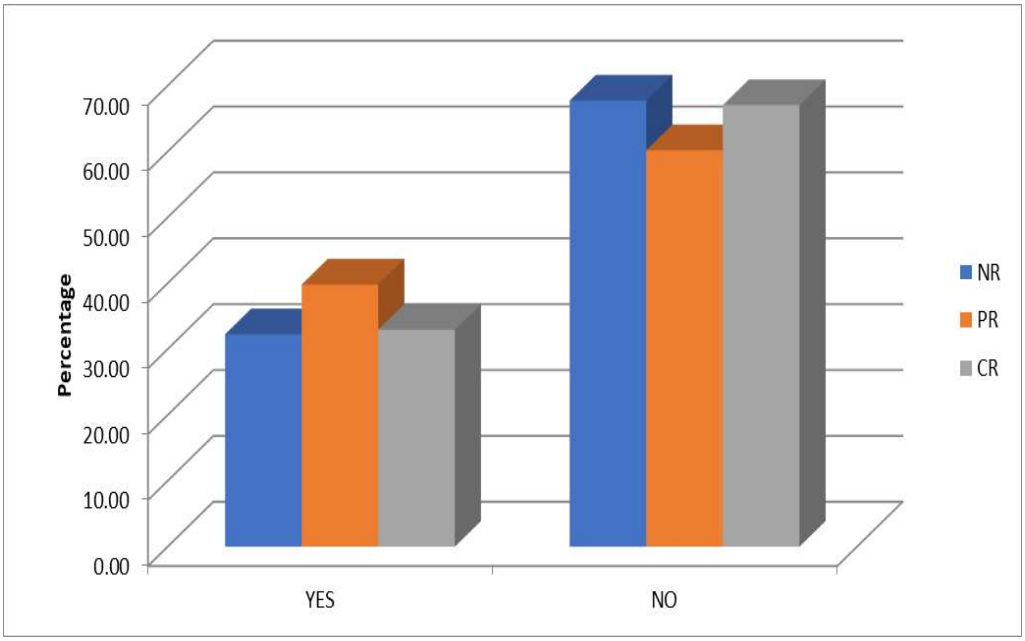


TABLE 62: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR TROPICAL ETIOLOGY OF AKI

	NR		PR		CR		NR+PR VS CR
	No.	%	No.	%	No.	%	p-value
TROPICAL	19	61.29	29	28.16	120	69.36	0.0001
NON-TROPICAL	12	38.71	74	71.84	53	30.64	

Patients with CA-AKI due to tropical etiology had much better chance of renal recovery in comparison to non-tropical etiologies. The difference was statistically significant with P value below 0.01.

FIGURE 79: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR TROPICAL ETIOLOGY OF AKI

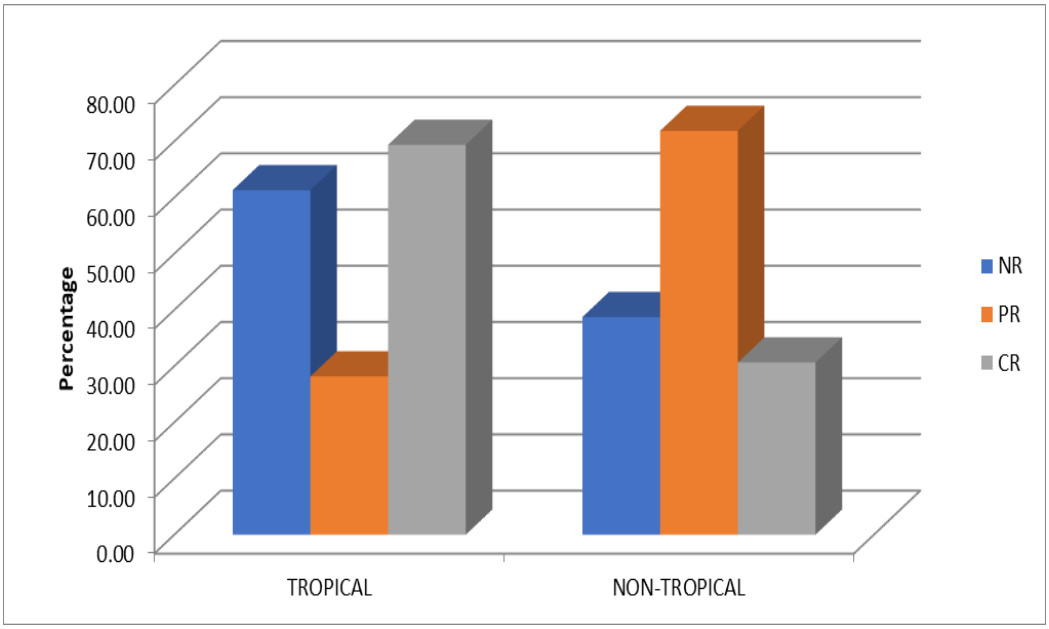


TABLE 63: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR TYPE OF AKI

	NR		PR		CR		NR+PR VS CR
TYPE OF AKI	No.	%	No.	%	No.	%	p-value
HYPOPERFUSION	3	9.68	12	11.65	118	68.21	0.0001
INTRINSIC	28	90.32	69	66.99	36	20.81	
OBSTRUCTIVE	0	0.00	22	21.36	19	10.98	

Intrinsic AKI etiologies were associated with significantly poor renal recovery in comparison to Prerenal (Hypoperfusion to begin with) and obstructive etiology and it was statistically significant with P value below 0.001. Obstructive AKI had many cases with partial recovery which was statistically more in comparison to prerenal.

FIGURE 80: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR TYPE OF AKI

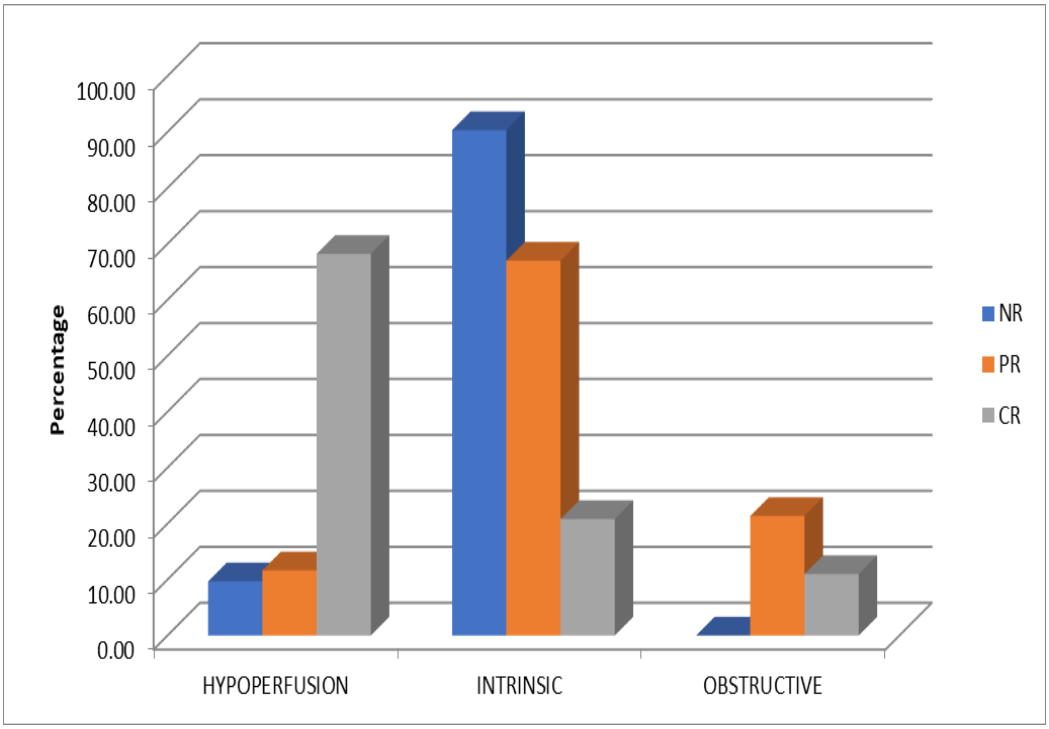


TABLE 64: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR STAGE OF AKI

	NR		PR		CR		NR+PR VS CR
Stage	No.	%	No.	%	No.	%	p-value
1	0	0.00	0	0.00	26	15.03	0.0001
2	7	22.58	29	28.16	99	57.23	
3	24	77.42	74	71.84	48	27.75	

All the KDIGO stage 1 AKI had complete recovery while 72 % of Stage 2 and only 32% of stage 3 had complete recovery and this difference was statistically significant.

FIGURE 81: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR STAGE OF AKI

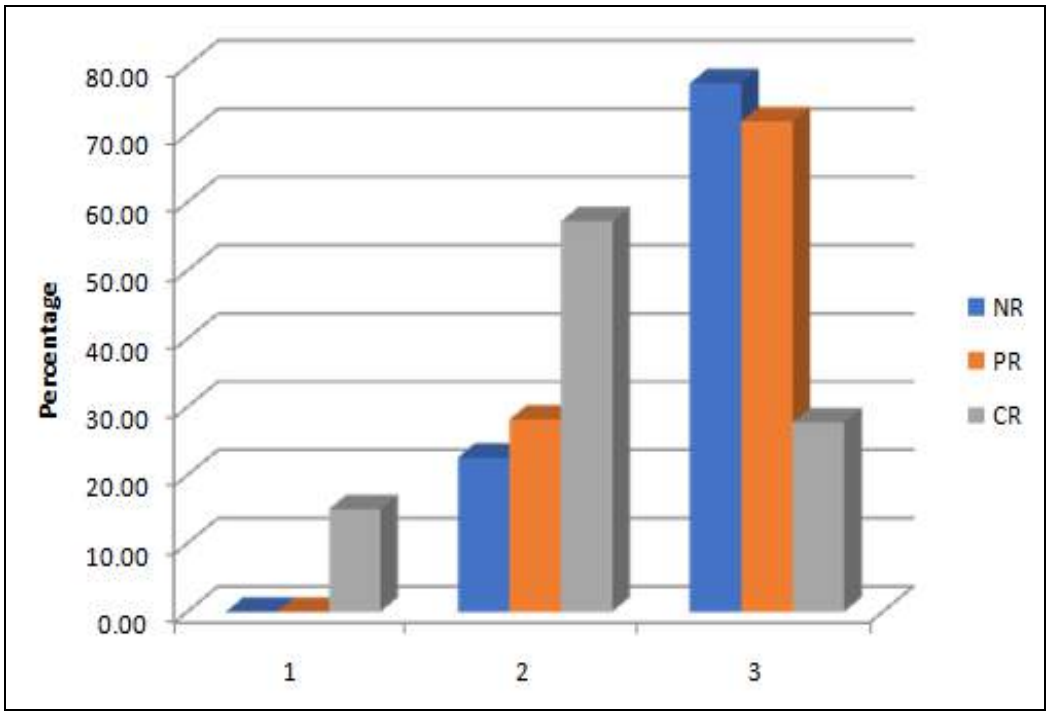


TABLE 65: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR VARIOUS ETILOGIES OF AKI

	NR		PR		CR		NR+PR VS CR
	No.	%	No.	%	No.	%	p-value
CARDIORENAL	0	0.00	12	11.65	1	0.58	0.003
CRUSH INJURY	0	0.00	4	3.88	8	4.62	0.7
DRUG	11	35.48	12	11.65	6	3.47	0.007
HYPOVOLUEMIA	1	3.23	4	3.88	66	38.15	<0.0001
POISONING	1	3.23	1	0.97	4	2.31	0.38
SEPSIS	8	25.81	34	33.01	24	13.87	0.002
SNAKE BITE	3	9.68	2	1.94	1	0.58	0.047
TROPICAL FEVER	2	6.45	7	6.80	33	19.08	0.001
U.O.	0	0.00	22	21.36	19	10.98	0.16
GLOMERULONEPHRITUS	2	6.45	3	2.91	3	1.73	0.3
MULTIFACTORIAL AKI	8	25.81	21	20.39	11	6.36	0.0001

Patients with CA-AKI etiologies like Cardiorenal syndrome, drug induced AKI, sepsis, snake bite, tropical fever and multifactorial AKI had less chance of complete renal recovery in comparison to patients without these etiologies. On contrary, patients with Hypovolemia had better chances of renal recovery in comparison to other etiologies taken together. Out of 5 survivors of pancreatitis 2 had complete recovery, both had BISAP score of 2 while rest 3 had partial recovery, 2 of them had BISAP score of 2 while 1 had above 3

FIGURE 82: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR VARIOUS ETILOGIES OF AKI

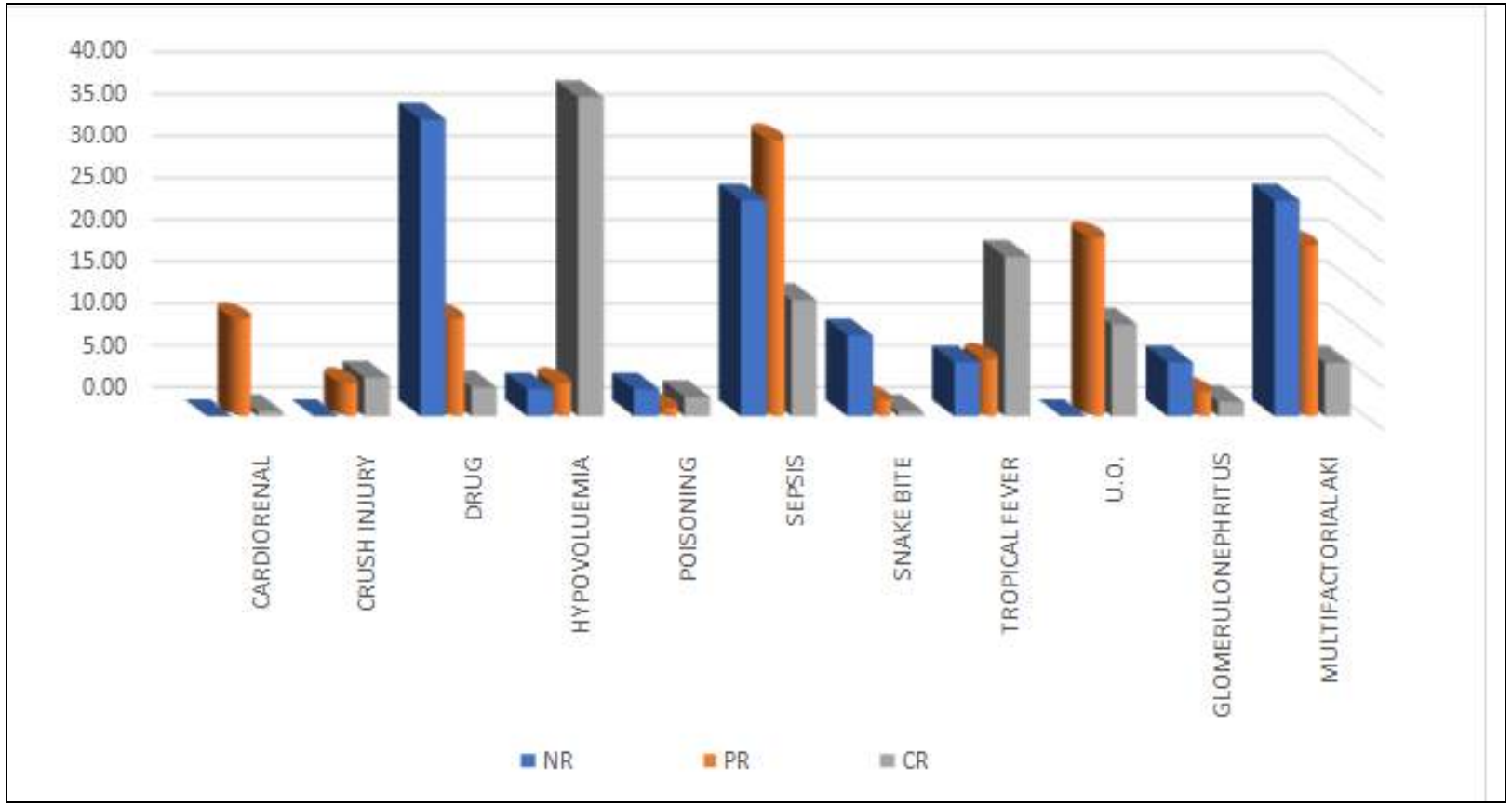


TABLE 66: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR MARKERS OF SEVERITY OF ILLNESS

	NR		PR		CR		NR+PR VS CR
	No.	%	No.	%	No.	%	p-value
NEED OF DIALYSIS	16	51.61	29	28.16	13	7.51	0.001
NEED OF ICU	10	32.26	39	37.86	42	24.28	0.019
OLIGURIA	16	51.61	29	28.16	21	12.14	0.0001
NEED OF INOTROPES	10	32.26	33	32.04	21	12.14	0.0001
MULTI ORGAN FAILURE	25	81	37	36	10	6	0.0001
NEED OF M. VENTILATION	16	51	33	32	7	4	0.0001

CA-AKI Patients requiring ICU stay, inotropic support, mechanical ventilation, dialysis, patients who had multiorgan failure, oliguria had significantly lesser chances of renal recovery in comparison to those without it suggesting that severity of underlying comorbidities, etiology and CA-AKI had a significant relation to outcome.

FIGURE 83: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR MARKERS OF SEVERITY OF ILLNESS

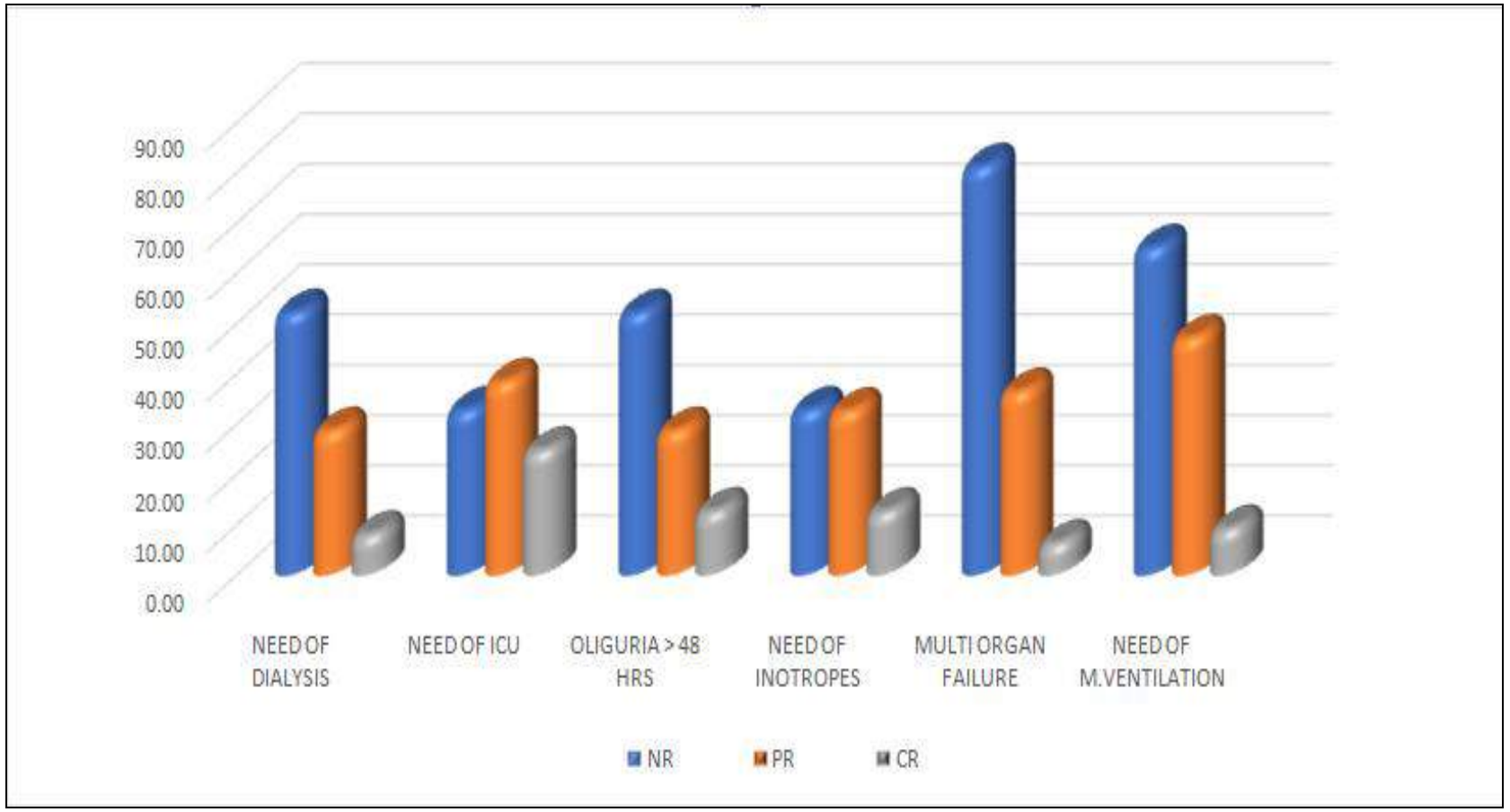


TABLE 67: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR MODE OF DIALYSIS

	NR		PR		CR		NR+PR VS CR
MODE OF DIALYSIS	No.	%	No.	%	No.	%	p-value
CRRT	0	0.00	1	3.45	1	7.69	0.48
HD	12	75.00	13	44.83	9	69.23	
PD	2	12.50	8	27.59	1	7.69	
SLED	2	12.50	7	24.14	2	15.38	

Though most the patients who completely recovered after requiring renal replacement therapy were of hemodiayis but it was statistically not significant. Overall various modes of dialysis had no significant impact on renal recovery with a p value of 0.48

FIGURE 84: COMPARING COMPLETE, PARTIAL AND NON-RECOVERY FOR MODE OF DIALYSIS

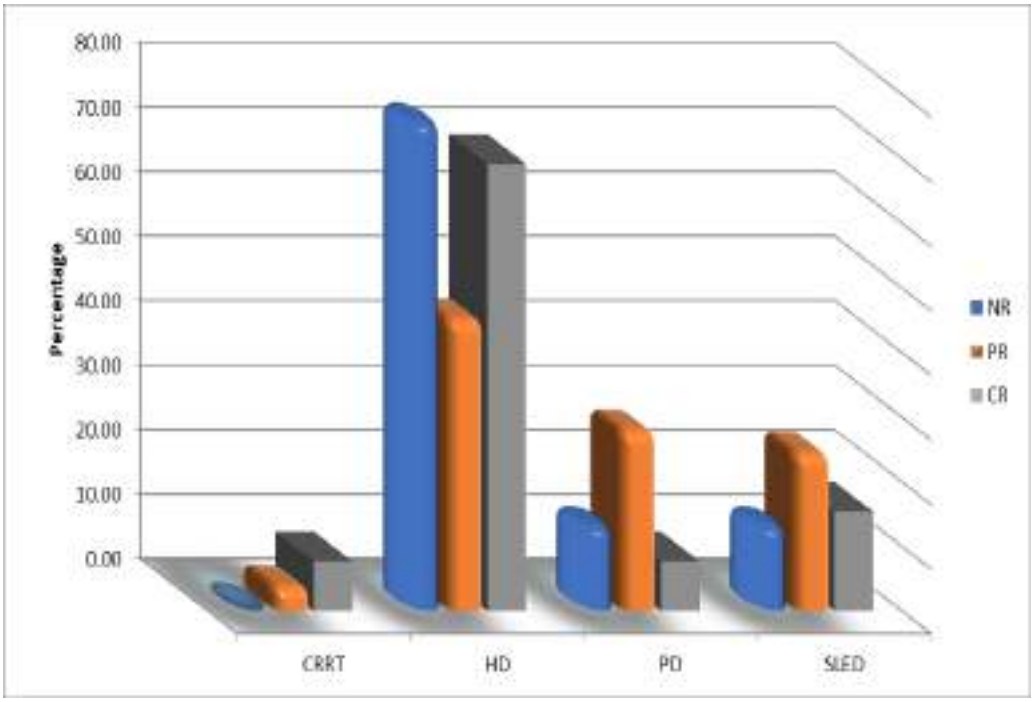


TABLE 68: VARIOUS FACTORS AFFECTING NEED OF ICU ADMISSION

	NEED OF ICU		NO NEED OF ICU		UNNIVARIATE	MULTIVARIATE
	NO.	%	NO	%	p-value	p-value
AGE GROUP						
<30 years	20	42.55	27	57.45		
30-39 years	23	43.40	30	56.60	0.1	NA
40-49 years	45	35.43	82	64.57		
50-59 years	43	40.95	62	59.05		
>60 years	28	58.33	20	41.67		
GENDER						
F	70	44.30	88	55.70	0.41	NA
M	89	40.09	133	59.91		
RESIDENTIAL STATUS						
RURAL	102	46.15	119	53.85	0.04	0.07
URBAN	57	35.85	102	64.15		
COMORBIDITIES						
NONE	60	36.81	103	63.19	0.08	NA
DM	35	57.38	26	42.62	0.007	0.3
HTN	48	40.34	71	59.66	0.68	NA
CKD	59	55.66	47	44.34	0.0006	0.03
CAD	18	64.29	10	35.71	0.01	0.9
CVA	11	57.89	8	42.11	0.15	NA
COAD	13	59.09	9	40.91	0.09	NA
CLD	20	66.67	10	33.33	0.002	0.01
NUMBER OF COMORBIDITIES						
NONE	60	36.81	103	63.19		
1	35	32.71	72	67.29	0.0001	0.09
2	34	45.33	41	54.67		
3 OR MORE	30	85.71	5	14.29		
ETIOLOGIES						
SEPSIS	60	60.00	40	40.00	0.00001	0.001
TROPICAL FEVER	32	58.18	23	41.82	0.007	0.2
HYPOVOLEMIA	11	15.07	62	84.93	0.00001	NA
DRUGS	4	11.43	31	88.57	0.0001	NA
CARDIORENAL	10	66.67	5	33.33	0.04	0.08

POISONING	13	86.67	2	13.33	0.0003	0.001
SNAKE BITE	5	62.50	3	37.50	0.23	NA
CRUSH INJURY	15	100.00	0	0.00	0.00001	0.001
GLOMERULONEPHRITIS	1	16.66	7	83.34	0.09	NA
OTHERS (HUS,HRS,TLS, MM, G6PD)	2	25.00	6	75.00	0.32	NA
URINARY OBSTRUCTION	3	7.32	38	92.68	0.0001	NA
PREECLAMSIA/ HELLP	3	42.86	4	57.14	0.95	NA
PATTERN OF AKI					0.16	NA
MED	118	40.83	171	59.17		
OBS	14	60.87	9	39.13		
SUR	27	39.71	41	60.29		
STAGE OF AKI					0.0001	0.4
1	0	0.00	26	100.00		
2	25	17.99	114	82.01		
3	134	62.33	81	37.67		
OLIGURIA	79	71	33	29	0.0001	0.12
NEED OF INOTROPES	84	74.34	29	25.66	0.0001	0.04
NEED OF DIALYSIS	97	78.86	26	21.14	0.0001	0.09
MODE OF DIALYSIS					0.0001	0.15
CRRT	8	100.00	0	0.00		
HD	24	48.00	26	52.00		
PD	28	100.00	0	0.00		
SLED	37	100.00	0	0.00		
OUTCOME						
CR	42	24.28	131	75.72	0.001	NA
PR	39	37.86	64	62.14	0.33	NA
NR	10	32.26	21	67.74	0.25	NA
7 DAY MORTALITY	39	95.12	2	4.88	0.0001	NA
30 DAY MORTALITY	68	93.15	5	6.85	0.0001	NA
READMISSION	50	52.08	46	47.92	0.01	NA

UNIVARIATE ANALYSIS- Need of ICU was significantly more in people from rural area, people with diabetes, underlying CKD, CAD and CLD. People with a greater number of comorbidities required ICU more frequently. People with sepsis, tropical fevers, cardiorenal syndrome, crush injury and poisoning had higher need of ICU stay, while patients with hypovolemia, drug induced AKI and urinary obstruction had much less need. Increasing stage of AKI, oliguria, need of dialysis and inotropes, dialysis modality other than HD, all were significantly associated with need of ICU. Those patients who recovered completely had much less need of ICU. Early as well as late mortality was significantly associated with need of ICU admission. Readmissions were also common for patients who required ICU admission.

MULTIVARIATE ANALYSIS- On multivariate analysis, underlying CKD, CLD, Sepsis, poisoning, crush injury and need of inotropic support were significantly associated with ICU admission.

TABLE 69: VARIOUS FACTORS AFFECTING NEED OF MECHANICAL VENTILATION

	NEED OF VENTILATION		NO NEED OF VENTILATION		UNIVARIATE	MULTIVARIATE
	NO.	%	NO.	%	p-value	p-value
AGE GROUP						
<30 years	17	36.17	30	63.83		
30-39 years	16	30.19	37	69.81	0.21	NA
40-49 years	31	24.41	96	75.59		
50-59 years	31	29.52	74	70.48		
>60 years	20	41.67	28	58.33		
GENDER						
F	51	32.28	107	67.72	0.47	NA
M	64	28.83	158	71.17		
RESIDENTIAL STATUS					0.6	NA
RURAL	71	32.13	150	67.87		
URBAN	44	27.67	115	72.33		
COMORBIDITIES						
NONE	36	22.09	127	77.91	0.002	NA
DM	30	49.18	31	50.82	0.0004	0.04
HTN	28	23.53	91	76.47	0.06	NA
CKD	40	37.74	66	62.26	0.04	0.08
CAD	12	42.86	16	57.14	0.13	NA
CVA	7	36.84	12	63.16	0.52	NA
COAD	12	54.55	10	45.45	0.01	0.09
CLD	13	43.33	17	56.67	0.1	NA
NUMBER OF COMORBIDITIES						
NONE	36	22.09	127	77.91		
1	24	22.43	83	77.57	0.0001	0.03
2	27	36.00	48	64.00		
3 OR MORE	28	80.00	7	20.00		
ETIOLOGIES						
SEPSIS	44	44.00	56	56.00	0.0004	0.02
TROPICAL FEVER	20	36.36	35	63.64	0.28	NA
HYPOVOLEMIA	7	9.59	66	90.41	0.00001	NA
DRUGS	3	8.57	32	91.43	0.0001	NA
CARDIORENAL	8	53.33	7	46.67	0.04	0.2

POISONING	12	80.00	3	20.00	0.0001	0.01
SNAKE BITE	5	62.50	3	37.50	0.2	NA
CRUSH INJURY	11	73.33	4	26.67	0.001	0.02
GLOMERULONEPHRITIS	0	0.00	8	100	0.00001	NA
OTHERS (HUS,HRS,TLS,MM,G6PD)	1	12.50	7	87.50	0.09	NA
URINARY OBSTRUCTION	1	2.44	40	97.56	0.0001	NA
PREECLAMSIA/HELLP	3	42.86	4	57.14	0.9	NA
PATTERN OF AKI					0.12	NA
MED	90	31.14	199	68.86		
OBS	10	43.48	13	56.52		
SUR	15	22.06	53	77.94		
STAGE OF AKI					0.0001	0.2
1	0	0	26	100		
2	8	5.76	131	94.24		
3	107	49.77	108	50.23		
OLIGURIA	70	63	42	38	0.0001	0.3
NEED OF INOTROPES	75	66.37	38	33.63	0.0001	0.03
NEED OF DIALYSIS	76	61.79	47	38.21	0.0001	0.1
MODE OF DIALYSIS					0.0001	0.1
CRRT	8	100.00	0	0.00		
HD	16	32.00	34	68.00		
PD	26	92.86	2	7.14		
SLED	26	70.27	11	29.73		
OUTCOME						
CR	12	6.94	161	93.06	0.0001	NA
PR	35	33.98	68	66.02	0.33	NA
NR	9	29.03	22	70.97	0.87	NA
7 DAY MORTALITY	37	90.24	4	9.76	0.0001	NA
30 DAY MORTALITY	59	80.82	14	19.18	0.0001	NA
READMISSION	39	40.63	57	59.38	0.01	NA

UNIVARIATE ANALYSIS- Need of ventilation was significantly more in people with diabetes, underlying CKD, and COAD. People with a greater number of comorbidities required ventilation more frequently. People with sepsis, cardiorenal syndrome, crush injury and poisoning had higher need of ventilation, while patients with hypovolemia, drug induced AKI, glomerulonephritis and urinary obstruction had much less need. Increasing stage of AKI, oliguria, need of dialysis and inotropes, dialysis modality other than HD, all were significantly associated with need of Ventilation. Those patients who recovered completely had much less need of ventilation. Early as well as late mortality was significantly associated with need of ventilation. Readmissions were also common for patients who required ventilation.

MULTIVARIATE ANALYSIS- On multivariate analysis, underlying diabetes, higher number of comorbidities, Sepsis, poisoning, crush injury and need of inotropic support were significantly associated with need of ventilation.

TABLE 70: VARIOUS FACTORE AFFECTING LENGTH OF HOSPITAL STAY

	Hospital stay			UNIVARIATE	MULTIVARIATE
	Mean	SD	NO.	P - value	p-value
OVERALL	9.1	5.04	380		
AGE GROUP					
<30 years	12.6	5.78	47	0.01	0.5
30-39 years	10.1	6.08	53	0.18	NA
40-49 years	8.2	4.54	127	0.08	NA
50-59 years	8.4	4.22	105	0.19	NA
>60 years	9.1	4.09	48	1.0	NA
GENDER					
F	8.75	5.04	158	0.46	NA
M	9.33	5.1	222		
RESIDENTIAL STATUS					
RURAL	9.57	5.51	221	0.14	NA
URBAN	8.82	4.3	159		
COMORBIDITIES					
NONE	9.6	5.8	163	0.31	NA
DM	9	3.77	61	0.88	NA
HTN	8.24	4.44	119	0.1	NA
CKD	9.08	4.07	106	0.97	NA
CAD	10.14	4.16	28	0.3	NA
CVA	9.6	4.1	19	0.67	NA
COAD	8.7	3.9	22	0.71	NA
CLD	8.86	4.69	30	0.80	NA
NUMBER OF COMORBIDITIES					
NONE	9.6	5.8	163	0.31	NA
1	8.4	4.5	107	0.19	NA
2	8.8	4.1	75	0.62	NA
3 OR MORE	8.9	4	35	0.81	NA
ETIOLOGIES					
SEPSIS	8.9	4.2	100	0.71	NA
TROPICAL FEVER	8.8	3.8	55	0.67	NA
HYPOVOLEMIA	5.5	3.9	73	0.0001	0.001
DRUGS	8.5	4.1	35	0.49	NA

CARDIORENAL	9	2.4	15	0.93	NA
POISONING	14.9	5.7	15	0.0001	0.03
SNAKE BITE	17.8	4.9	8	0.0001	0.02
CRUSH INJURY	18	4.8	15	0.0001	0.02
GLOMERULONEPHRITIS	10.7	2.1	8	0.37	NA
URINARY OBSTRUCTION	9.07	3.5	41	0.97	NA
PREECLAMSIA/ HELLP	8.2	3.9	7	0.63	NA
PATTERN OF AKI					
MED	8.56	4.7	289	0.15	NA
OBS	9.3	5.5	23	0.85	NA
SUR	11.25	5.5	68	0.0015	0.34
STAGE OF AKI					
1	3.7	1	26	0.0001	0.45
2	7.1	3.1	139	0.0001	0.12
3	11	5.38	215	0.0001	0.09
NEED OF ICU	11.3	5.6	159	0.0001	0.07
NEED OF VENTILATION	13.5	5.3	115	0.0001	0.04
OLIGURIA	12.6	5.9	112	0.0001	0.1
NEED OF INOTROPES	11.9	5.3	113	0.0001	0.01
NEED OF DIALYSIS	11.1	5.9	123	0.0003	0.03
MODE OF DIALYSIS					
CRRT	10.2	7.7	8	0.54	NA
PD	8.2	5.3	28	0.36	NA
SLED	9.2	5.4	37	0.90	NA
HD	14.06	4.9	50	0.0001	0.23
OUTCOME					
CR	7.59	4.67	173	0.001	0.34
PR	10.5	3.7	103	0.01	0.07
NR	13.03	5.09	31	0.0001	0.02
30 DAY MORTALITY	8.89	6	73	0.75	NA

UNIVARIATE ANALYSIS- Length of Hospital stay was significantly longer in comparison overall study population in patients with age below 30, in patients with poisoning, snake bite and crush injury. Patients with surgical AKI also had longer stay while patients with hypovolemia, stage 1 and 2 of AKI had relatively shorted stay. Stage 3 AKI, oliguria, need of dialysis, ICU, ventilation and inotropes, all had significantly longer hospital stay. Those patients who recovered completely had much shorter hospital stay. Patients with partial or no recovery and need of hemodialysis (HD) was associated with prolonged stay.

MULTIVARIATE ANALYSIS- On multivariate analysis comparing multiple means using ANOVA, patients with poisoning, crush injury, snake bite, need of inotropes, ventilation, and dialysis had significantly longer hospital stay. Longer stay was significantly associated with Non-recovery.

TABLE 71: VARIOUS FACTORS AFFECTING NEED OF DIALYSIS

	NEED OF DIALYSIS		NO NEED OF DIALYSIS		UNIVARIATE	MULTIVARIATE
	NO.	%	NO.	%		
AGE GROUP						
<30 years	13	27.66	34	72.34		
30-39 years	14	26.42	39	73.58	0.13	NA
40-49 years	38	29.92	89	70.08		
50-59 years	35	33.33	70	66.67		
>60 years	23	47.92	25	52.08		
GENDER						
F	50	31.65	108	68.35	0.8	NA
M	73	32.88	149	67.12		
RESIDENTIAL STATUS					0.58	NA
RURAL	74	33.48	147	66.52		
URBAN	49	30.82	110	69.18		
COMORBIDITIES						
NONE	40	24.54	123	75.46	0.004	NA
DM	33	54.10	28	45.90	0.0001	0.24
HTN	30	25.21	89	74.79	0.04	0.78
CKD	43	40.57	63	59.43	0.03	0.04
CAD	12	42.86	16	57.14	0.21	NA
CVA	8	42.11	11	57.89	0.35	NA
COAD	11	50.00	11	50.00	0.07	NA
CLD	16	53.33	14	46.67	0.01	0.15
NUMBER OF COMORBIDITIES						
NONE	40	24.54	123	75.46		
1	31	28.97	76	71.03	0.0001	0.02
2	28	37.33	47	62.67		
3 OR MORE	24	68.57	11	31.43		

ETIOLOGIES						
SEPSIS	53	53.00	47	47.00	0.0001	0.03
TROPICAL FEVER	13	23.64	42	76.36	0.13	NA
HYPOVOLEMIA	4	5.48	69	94.52	0.0001	NA
DRUGS	7	20.00	28	80.00	0.1	NA
CARDIORENAL	3	20.00	12	80.00	0.29	NA
POISONING	10	66.67	5	33.33	0.003	0.06
SNAKE BITE	8	100.00	0	0.00	0.0001	0.01
CRUSH INJURY	8	53.33	7	46.67	0.075	NA
GLOMERULONEPHRITIS	4	50.00	4	50.00	0.28	NA
OTHERS	5	62.50	3	37.50	0.7	NA
URINARY OBSTRUCTION	5	12.20	36	87.80	0.003	NA
PREECLAMSIA/HELLP	3	42.86	4	57.14	0.3	NA
PATTERN OF AKI					0.68	NA
MED	94	32.53	195	67.47		
OBS	9	39.13	14	60.87		
SUR	20	29.41	48	70.59		
NEED OF ICU	97	61	62	39	0.0001	0.09
NEED OF VENTILATION	76	66.09	39	33.91	0.0001	0.1
NEED OF INOTROPES	76	67.26	37	32.74	0.0001	0.03
OUTCOME						
CR	13	7.51	160	92.49	0.0001	NA
PR	29	28.16	74	71.84	0.28	NA
NR	16	51.61	15	48.39	0.01	NA
7 DAY MORTALITY	39	95.12	2	4.88	0.0001	NA
30 DAY MORTALITY	65	89.04	8	10.96	0.0001	NA
READMISSION	54	56.25	42	43.75	0.0001	NA

UNIVARIATE ANALYSIS- Need of dialysis was significantly more in people with diabetes, hypertension, underlying CKD, and CLD. People with a greater number of comorbidities required dialysis more frequently. People with sepsis, poisoning and snake bite had higher need of dialysis, while patients with hypovolemia, and urinary obstruction had much less need. Need of ICU, ventilation and inotropes were significantly associated with need of dialysis. Those patients who recovered completely had much less need of dialysis while more than 50% Non-recovered requiring dialysis. Early as well as late mortality was significantly associated with need of dialysis. Readmissions were also common for patients who required dialysis.

MULTIVARIATE ANALYSIS- On multivariate analysis, underlying CKD, higher number of comorbidities, Sepsis, snake bite, and need of inotropic support were significantly associated with need of dialysis.

TABLE 72: VARIOUS FACTORS AFFECTING EARLY MORTALITY (7 DAY)

	7 DAY MORTALITY (EARLY MORTALITY)					
	YES		NO		UNIVARIATE	MULTIVARIATE
	NO.	%	NO.	%		
AGE GROUP					0.47	NA
<30 years	2	4.26	45	95.74		
30-39 years	4	7.55	49	92.45		
40-49 years	16	12.60	111	87.40		
50-59 years	13	12.38	92	87.62		
>60 years	6	12.50	42	87.50		
GENDER					0.51	NA
F	19	12.03	139	87.97		
M	22	9.91	200	90.09		
RESIDENTIAL STATUS					0.53	NA
RURAL	22	9.95	199	90.05		
URBAN	19	11.95	140	88.05		
COMORBIDITIES						
NONE	11	6.75	152	93.25	0.02	NA
DM	13	21.31	48	78.69	0.003	0.75
HTN	19	15.97	100	84.03	0.02	0.42
CKD	22	20.75	84	79.25	0.0001	0.15
CAD	3	10.71	25	89.29	0.98	NA
CVA	3	15.79	16	84.21	0.47	NA
COAD	4	18.18	18	81.82	0.24	NA
CLD	8	26.67	22	73.33	0.003	0.09
NUMBER OF COMORBIDITIES						
NONE	11	6.75	152	93.25	0.0001	0.02
1	7	6.54	100	93.46		
2	14	18.67	61	81.33		
3 OR MORE	9	25.71	26	74.29		
ETIOLOGIES						
SEPSIS	31	31.00	69	69.00	0.0001	0.001

TROPICAL FEVER	6	10.91	49	89.09	0.92	NA
HYPOVOLEMIA	1	1.37	72	98.63	0.0001	NA
DRUGS	1	2.86	34	97.14	0.11	NA
CARDIORENAL	0	0.00	15	100.00	0.0001	NA
POISONING	0	0.00	15	100.00	0.0001	NA
SNAKE BITE	0	0.00	8	100.00	0.0001	NA
CRUSH INJURY	0	0.00	15	100.00	0.0001	NA
GLOMERULONEPHRITIS	0	0.00	8	100.00	0.0001	NA
OTHERS	2	25.00	6	75.00	0.4	NA
URINARY OBSTRUCTION	0	0.00	41	100.00	0.0001	NA
PREECLAMSIA/HELLP	0	0.00	7	100.00	0.0001	NA
PATTERN OF AKI						
MED	33	11.42	256	88.58	0.23	NA
OBS	4	17.39	19	82.61		
SUR	4	5.88	64	94.12		
STAGE OF AKI						
1	0	0	26	100	0.0001	0.5
2	1	0.72	138	99.28		
3	40	18.60	175	81.40		
NEED OF ICU	39	25	120	75	0.0001	0.08
OLIGURIA	19	17	93	83	0.0001	0.13
NEED OF INOTROPES	39	34.51	74	65.49	0.0001	0.001
NEED OF DIALYSIS	39	31.71	84	68.29	0.0001	0.001
NEED OF M. VENTILATION	37	32.17	78	67.83	0.0001	0.12
MODE OF DIALYSIS						
CRRT	4	50.00	4	50.00	0.0001	0.24
HD	0	0.00	50	100.00		
PD	17	60.71	11	39.29		
SLED	18	48.65	19	51.35		

UNIVARIATE ANALYSIS- Early mortality was significantly more in people with diabetes, hypertension, underlying CKD, and CLD. People with a greater number of comorbidities had higher early mortality. People with sepsis had significantly higher early mortality. Increasing stage of AKI, oliguria, need of ICU, dialysis, Ventilation, inotropes, dialysis modality other than HD, all were significantly associated with higher early mortality.

MULTIVARIATE ANALYSIS- On multivariate analysis, higher number of comorbidities, Sepsis, need of dialysis and need of inotropic support were significantly associated with early mortality.

TABLE 73: VARIOUS FACTORS AFFECTING 30 DAY MORTALITY

	30 DAY MORTALITY (OVERALL MORTALITY)					
	YES		NO		UNIVARIATE	MULTIVARIATE
	NO.	%	NO.	%		
AGE GROUP					0.9	NA
<30 years	9	19.15	38	80.85		
30-39 years	10	18.87	43	81.13		
40-49 years	27	21.26	100	78.74		
50-59 years	17	16.19	88	83.81		
>60 years	10	20.83	38	79.17		
GENDER					0.92	N.A.
F	30	18.99	128	81.01		
M	43	19.37	179	80.63		
RESIDENTIAL STATUS					0.68	N.A.
RURAL	44	19.91	177	80.09		
URBAN	29	18.24	130	81.76		
COMORBIDITIES						
NONE	25	15.34	138	84.66	0.001	NA
DM	20	32.79	41	67.21	0.001	0.74
HTN	33	27.73	86	72.27	0.004	0.13
CKD	36	33.96	70	66.04	<0.00001	0.04
CAD	6	21.43	22	78.57	0.75	NA
CVA	7	36.84	12	63.16	0.04	0.23
COAD	5	22.73	17	77.27	0.66	NA
CLD	13	43.33	17	56.67	<0.001	0.03
NUMBER OF COMORBIDITIES					0.001	0.03
NONE	25	15.34	138	84.66		
1	11	10.28	96	89.72		
2	20	26.67	55	73.33		
3 OR MORE	17	48.57	18	51.43		
ETIOLOGIES						

SEPSIS	34	34.00	66	66.00	<0.0001	0.03
TROPICAL FEVER	13	23.64	42	76.36	0.36	NA
HYPOVOLEMIA	2	2.74	71	97.26	<0.0001	NA
DRUGS	6	17.14	29	82.86	0.74	NA
CARDIORENAL	2	13.33	13	86.67	0.56	NA
POISONING	9	60.00	6	40.00	<0.0001	0.01
SNAKE BITE	2	25.00	6	75.00	0.65	NA
CRUSH INJURY	3	20.00	12	80.00	0.93	NA
GLOMERULONEPHRITIS	0	0.00	8	100.00	0.0001	NA
OTHERS	2	25.00	6	75.00	0.45	NA
URINARY OBSTRUCTION	0	0.00	41	100.00	0.0001	NA
PREECLAMSIA/ HELLP	0	0.00	7	100.00	0.0001	NA
PATTERN OF AKI					0.2	NA
MED	61	21.11	228	78.89		
OBS	4	17.39	19	82.61		
SUR	8	11.76	60	88.24		
STAGE OF AKI					0.001	0.35
1	0	0	26	100		
2	4	2.88	135	97.12		
3	69	32.09	146	67.91		
NEED OF ICU	68	43	91	57	0.001	0.12
OLIGURIA	46	41	66	59	0.001	0.1
NEED OF INOTROPES	49	43.36	64	56.64	0.001	0.01
NEED OF DIALYSIS	65	52.85	58	47.15	0.001	0.01
NEED OF M. VENTILATION	59	51.30	56	48.70	0.01	0.1
MODE OF DIALYSIS						
CRRT	6	75.00	2	25.00	0.001	0.13
HD	16	32.00	34	68.00		
PD	17	60.71	11	39.29		
SLED	26	70.27	11	29.73		

UNIVARIATE ANALYSIS- 30-day mortality was significantly more in people with diabetes, hypertension, underlying CKD, CVA and CLD. People with a greater number of comorbidities had higher mortality. People with sepsis and poisoning had significantly higher mortality. Increasing stage of AKI, oliguria, need of ICU, dialysis, Ventilation, inotropes, dialysis modality other than HD, all were significantly associated with higher mortality.

MULTIVARIATE ANALYSIS- On multivariate analysis, underlying CKD and CLD, higher number of comorbidities, Sepsis, poisoning, need of dialysis and need of inotropic support were significantly associated with mortality.

DISCUSSION

Community-acquired AKI (CA-AKI) is a common, previously unrecognized, potentially preventable disease especially in developing countries like ours which need attention not just by the clinicians but also by the epidemiologists and requires prevention at grass-root level. Because the causative factors of community-acquired AKI originate in the communities, environmental and lifestyle differences potentially affect the incidence and patterns of CA-AKI. Therefore, uncovering the local features of community-acquired AKI is necessary for formulating site-specific preventive and therapeutic manoeuvres. With limited data, available especially from this part of country, we did this study to understand the epidemiology and outcome of CA-AKI in our region.

DEMOGRAPHIC FEATURE AND BASELINE CHARATERSTICS

We prospectively studied 380 patients after screening 54285 patients with overall 7 patients per 1000 admission having CA-AKI. These results were similar to study done recently by Vikrant S. et al[82] though our study also included patients with underlying CKD. Similar Data on CA-AKI from the developing world is scarce, had different definitions of AKI in different regions.[7] The incidence in international literature varies from 1% to 31%, and mortality ranges from 28% to 82% depending on the definition used.[87-89]

The proportion of patients with CA-AKI in our study was higher than those is relatively old studies.[72,78,79,86] Incidence of AKI has increased with time probably due to better identification, documentation and application of newer validated definitions.

Age range of the study subjects varied from 18 years to 69 years with a mean age of 45.4 years. It was comparable to as reported previously but relatively on the higher side.[5,41] We excluded age above 70 to avoid ageing related confounders. We also excluded pediatric patients. This mean age of AKI is still much lower than the mean age in western population [5,64] even for etiologies not limited to tropical climate like sepsis. Only 12% were more than 60 years of age which was 9% in previous study.[72] This may be reasoned by the shorter life expectancy

in Indian population and also implies to the occurrence of CAAKI in the younger age group as compared with developed countries. In a country like India, where access to health care insurance is limited, CAAKI in the middle-aged population creates a huge economic burden on families.

158 female patients out of 23209 (6.8 per 1000 admission) screened had CA-AKI with mean age of 43.8 years while 222 male patients out of 31076 (7.1 per 1000 admission) screened had CA-AKI with mean age of 46.6 years. Male to female ratio in CA-AKI was 1.4 :1. There was no significant gender difference in proportion of CA-AKI cases. More number of male seeking health care and having CA-AKI may be due to social construct of population or due to more exposure to diseases in males due to more outdoor activity.

58% patients came from rural background. It gives us an indirect estimate of delay in diagnosis and treatment of AKI and their illness compounded by indigenous treatment. They were relatively younger with less comorbidity, though both not statistically significant, had statistically more need of ICU stay on univariate analysis, had longer hospital stay (not statistically significant) and had higher rate of non or partial recovery (not statistically significant). Though these results were statistically not significant, but they were clinically relevant and highlight the need of care at peripheral and rural level.

Our study subjects seem to arise from 2 different populations, one population relatively young with no or single comorbidity at risk of tropical etiologies while another population with comorbidities complicated by non-tropical etiologies. 203 cases accounting for 53.4% of total cases were related to etiologies specific to tropical areas. This subgroup had mean age of 41.3 years, significantly less than the other subgroup (Non-tropical AKI with mean age 48.6 years) with 177 cases. Mean age of 48.6 years for non-tropical AKI etiologies is much worse than that in the western world (reported to be around 65-70 years).[5,64] This can be due to poor control of underlying disease, more intercurrent illnesses, intake of over-the-counter painkillers and indigenous medications.

Our study group had high rate of comorbidities. Hypertension was present in 31% of cases while diabetes was present in 16% (61) cases. 26 diabetic patients had evidence of microvascular complications. 13% patient had known chronic kidney disease while 15% more patients were diagnosed to have CKD after evaluation during study period. 6.57% patient had both diabetes and hypertension. 7.4% patients were known to have coronary artery disease while 5% had history of stroke. 5.8% had chronic obstructive airway disease and 7.9% had underlying chronic liver disease. 2.4% patients had history of treated and active malignancy. 3.7% had active or treated tuberculosis and 3.7% had nephrolithiasis. 5 patients had rheumatoid arthritis, 7 had positive serology for HCV, 2 for HBV and HIV each. 42% patients didn't have any comorbidity. Patients with DM, HTN, RA. CAD, CVA, CKD, COAD, CLD and Malignancy had their mean age above 50 with maximum for malignancy (59 years) and CAD (58 years).

With increasing age, the number of co-morbidities increased and this was statistically significant with p value below 0.01. Majority of the people with age above 50 years who developed AKI had 1 or more comorbidity. There is contrasting difference between mean age of patients with and without comorbidities. Mean age without comorbidities was 37 years while mean age with even one comorbidity was above 50 years. This was statistically significant using one-way AVONA for group with no comorbidity vs all the other groups separately (p-value below 0.01). There was no difference between other three groups.

Comparison of prevalence of various co morbid conditions in patients with CAAKI revealed approximately equal proportions of such diagnoses as diabetes, hypertension, heart disease and cancer in those previous studies which included underlying CKD patients[70,71] but higher than those which excluded underlying CKD.[72,81,82] In study by kaaviya et al[81], which excluded CKD, 10% were hypertension at admission, 17% had diabetes, 1.6% had Hepatitis B and HIV each. Rest comorbidities weren't described. In study by Anupama kaul et al[72], diabetics and CKD were excluded. In the study by Vikrant et al[82]

which also excluded CKD, 10% were hypertensive, 9% were diabetic, 9% had CAD, 14% COAD, 9% had CLD, 3% malignancies and tuberculosis. In the study by Goswami et al, which included CKD patients, 18% had hypertension, 19% diabetes, 14% were CKD, 7% CAD, 9% malignancy. In a systematic review and meta-analysis of prevalence, awareness, and control of hypertension in India by Anchala R et al[90], overall prevalence of hypertension in general population was 29.8%. This was quite close to 31% of hypertension in our cases suggesting that our subjects probably truly represent the population.

These CA-AKI patients with comorbidities represents the part of population who may benefit from more frequent blood tests in the event of an acute illness or medication change to screen early AKI and early institution of care.

Though there were overlaps between various types of AKI, overall 47.6% cases of AKI were classified primarily as intrinsic AKI based on clinical assessment and it was the most common type of AKI. It was followed by 41.6% cases of Pre-renal with clinical assessment consistent with renal hypoperfusion at the beginning of illness. 41 cases accounting for 10.8% cases were due to urinary obstruction (Post-renal). Relative a smaller number of prerenal cases than expected is probably due to the design of study being a tertiary care centre study hence missing large number of cases either managed at primary or secondary health facilities or by alternative medicine.

Most of our patients were having stage 3 AKI with 56.6% cases followed by stage 2 AKI accounting for 36.6% cases. Only 6.8% cases were diagnosed as stage 1 AKI.

The scanty number of stage 1 AKI can be due to delay in getting medical attention or due to referral bias.

ETIOLOGICAL SPECTRUM OF CA-AKI

In our study, 289 patients out of 31324 (9.2 cases per thousand admission) had CA-AKI related to medical causes accounting for 76.1 % cases, 68 had CA-AKI in surgical group out of 16311 (4.2 cases per thousand admission) and accounted for 17.9% cases and 23 cases in obstetric group out of 6650 cases (3.45 cases per thousand admission)

accounting for 6.1% cases of CA-AKI. Mean age of medical and surgical group was comparable (46.9 and 45.2 years) while that of obstetric group was 28.3 years.

Previous studies have shown that medical, surgical and obstetrical causes accounted for 77.5%–87.6%, 8.3%–8.9%, and 3.4%–14.2% cases, respectively.[56,57,72] Our observation thus stands nearly comparable to the previous Indian data with relatively higher number of Surgical- AKI.

Spectrum on primary pathologies which lead to AKI in medical cases was vast with Sepsis being most common cause (26%) followed by followed by hypovolemia (23.9%), tropical fever (19.0%), drugs (12.1%), poisoning (5.2%), cardiorenal (5.2%) snake bite 2.8% and glomerulonephritis (2.8%). HUS, HRS, TLS, G6PD and Myeloma accounted for 2.4% of total cases.

Urinary obstruction was the most common surgical causes of AKI (60.3%) with main etiologies being Nephrolithiasis and BPH. Crush injury (22.1) & sepsis (17.6%) accounted for remaining cases with most common site of sepsis being diabetic foot.

48% of obstetric cases were due to sepsis, 26% were a part of preeclampsia, rests were due to Hemorrhage, HELLP and HUS.

Sepsis -AKI

Sepsis, which was previously mainly associated with HA-AKI, was the most common etiology overall in CA-AKI also in our study accounting 26% of the total patients, 77% cases were medical, 12% were surgical while 11% cases were puerperal sepsis.

The change in trend in etiology of CA-AKI with increasing incidence of sepsis can be observed by comparing the past studies. Though many studies solely didn't study CA-AKI, but they had significant number of CA-AKI cases. Chugh et al[56] as well as Prakash J et al[86] showed the increasing incidence of sepsis causing AKI which was reflected later by many studies. In the study by Anupama kaul et al[72], ~ 14% patients of CA-AKI had Sepsis. In the study by Eswarappa M et al[69] with majority patients being CA-AKI had 38% cases due to sepsis. In the study by kaaviya et al[81], only 12% had sepsis as majority of their cases were

due to snake bite. In the study by vikrant et al[82], 53% cases were due to sepsis. In the studies by Aggarwal et al[76], kohli et al[78], and kumar et al[79], sepsis was the leading cause of AKI. Recent data from Chennai also show that sepsis is emerging as the most common medical cause of AKI.[91]

Increase in Sepsis-AKI represents AKI also actually highlight the fall in number of cases of tropical etiologies causing CA-AKI. Increased Sepsis-AKI cases can also be due to economics bias with less privileged ones not able to reach tertiary care centre.

The most common source of sepsis in our study was the lungs (31%) followed by pyelonephritis (21%). Lung as the focus of sepsis was in ~24% of cases in the study by Goswami et al[71] while it was 9% in the study by vikrant et al[82], both the studies considered tropical infections and sepsis together. Murugan et al reported AKI in 34% of patients with community-acquired pneumonia and AKI was also found to be common in non-severe categories.[92] Few patients had AKI associated with sepsis secondary to H1N1 influenza similar to Vikrant et al.[82] H1N1-infected patients involves multiple mechanisms, including hemodynamic instability, hypoxia, and probably direct effect of the H1N1 virus on the kidney.

Pyelonephritis was another major cause of sepsis associated AKI in our study with 21% cases of sepsis and 5% of total cases. Previous studies from various part of India and abroad have reported variable association of Pyelonephritis as a cause of CA-AKI1[81,82,93,94] In the 2011 study from Yemen[94], only 0.5% of patients with AKI was due to pyelonephritis. Even among Indian studies, few have highlighted pyelonephritis as an important cause of CAAKI. Recent studies by kaaviya et al[81] had pyelonephritis as 17% of total cases and second most common cause overall. vikrant et al[82] had 14% cases of AKI due to pyelonephritis. Dhanapriya et al[91] from Chennai in their analysis of 121 pyelonephritis patients admitted over 22 months, 42.9% were AKI stage 3, and 67% were diabetics. In our data of 21 patients with pyelonephritis, 13 were diabetic and rest were having underlying CKD. It appears that acute pyelonephritis is emerging as an important cause

of CAAKI in our region, afflicting those patients with predisposing factor like diabetes and CKD. It is disconcerting that multidrug resistant bacteria are the predominant organisms, possibly resulting from rampant and indiscriminate use of antibiotics in the community. UTI may cause sudden deterioration in renal function. Further, hypovolemia, hypotension, sepsis, the use of nephrotoxic drugs, contrast media and urinary obstruction are AKI risk factors in UTI patients.

Sepsis with gastroenteritis as a focus caused CA-AKI in 10% of sepsis-AKI cases in our study. 7% cases of Sepsis-AKI were due to infected diabetic foot while 5% were due to SBP. 2 cases were due to meningitis, 2 septic arthritis, 1 case each due to infective endocarditis, cholangitis and colitis. In 3 cases focus could not be identified. There is paucity of literature on such data in CA-AKI in previous studies. Vikrant et al[82] reported 2.6% of total cases due to infected diabetic foot, 1.6% due to viral hepatitis, 1% due SBP and 1 % due febrile neutropenia. Our study didn't had cases of viral hepatitis with AKI.

Tropical infections- AKI

Arthropod or water borne tropical infections are common in this part of world with seasonal variation. AKI has been described in association with a large number of tropical infections. The usual presentation is an acute febrile illness with severe headache, myalgia and involvement of other organ systems to a variable extent; jaundice, intravascular haemolysis and thrombocytopenia often accompany these early symptoms. Associated metabolic disturbances include lactic acidosis, hypoglycaemia, hyponatraemia and hyperkalaemia. However, the similarity of the clinical presentations of the various forms of infection-associated AKI makes identifying the different underlying tropical infections on the basis of clinical features alone difficult. Timely diagnosis, therefore, requires a good knowledge of the local epidemiology of community-acquired AKI and the availability of sensitive and diagnostic tests for specific pathogens.

In our study, Dengue fever was the most common tropical infection (44% of tropical infections) associated with AKI mainly in patients with

severe dengue. Scrub typhus was the 2nd most common cause (22%) followed by enteric fever (16%), malaria (9%) and leptospirosis (9%).

Dengue disease has emerged globally as the most frequent and medically relevant viral infection transmitted by a mosquito bite. Elevated temperature, rainfall and air humidity, as well as urban structures without adequate sanitation, are factors that contribute to the active dispersion of the mosquito and the dissemination of the various virus serotypes. Several forms of renal involvement have been identified in patients with dengue, including acute tubular necrosis, hemolytic uremic syndrome, proteinuria, glomerulopathy and nephrotic syndrome.[95] AKI is a significant, albeit poorly studied, complication of dengue. The data available are heterogeneous and mostly originate from retrospective case series and case reports. The reported frequency of this association exhibits wide variation in accordance to the particular population being assessed, severity of dengue, criteria used for the diagnosis of AKI and time of evaluation. Our study had 44% cases of tropical infections due to dengue (6% of total cases). Laoprasopwattana et al[96] reported an incidence of 0.9% among children in Thailand, and Lee et al[97] reported an incidence of 3.3% among adults in Taiwan. In a Brazilian intensive care unit for infectious diseases, dengue was the cause of 4% of the cases of AKI diagnosed using the RIFLE criteria.[98] In a study that employed AKIN criteria for diagnosis, the incidence of AKI was 10.8%.[99] Using the AKIN criteria in a retrospective analysis, Khalil et al[100] identified AKI in 13.3% of a series of patients with dengue confirmed by the presence of IgM antibodies, independent of the severity of disease; 64.8% of the patients were in Stage 1, 18.3% Stage 2 and 16.9% Stage 3 of the disease. In another study, the RIFLE classification was used to investigate the occurrence of AKI in patients with tropical acute febrile disease. The results showed that the incidence of AKI among patients with dengue upon admission to the hospital was 35.7%.[58] Several mechanisms have been proposed to account for the etiopathogenesis of dengue fever-induced AKI, including direct action by the virus, hemodynamic instability, rhabdomyolysis, hemolysis and acute

glomerular injury[101], often two or more mechanisms coexist simultaneously in the same patient.

Scrub typhus is a systemic disease that causes a generalized vasculitis in small blood vessels. The variable clinical manifestations may be correlated with the variant pathologic changes ranging from focal to disseminated multiorgan involvement. Reported severe clinical manifestations or complications of scrub typhus include meningoencephalitis, myocarditis, pneumonia, ARDS, AKI, GI bleeding, septic shock, and multiple organ failures which may be potentially fatal if there is a delay in diagnosis and treatment.[102-104] Scrub typhus is an important cause of acute undifferentiated febrile illnesses in the Indian subcontinent and it should be part of the differential diagnosis of acute febrile illness with AKI. In contrast with previous reports, involvement of the kidneys in scrub typhus seems to be relatively common. In recent studies AKI has been reported to occur in 9–40% of patients with scrub typhus.[58,102,103,105-107] AKI in scrub typhus is believed to be multifactorial in origin, the presumed causes being renal hypoperfusion resulting from shock or hypovolemia, vasculitis, rhabdomyolysis, acute interstitial nephritis, and direct microbial invasion of the renal tubules causing acute tubular necrosis.[108-110] In our study 22% of tropical infections (3% of total) were due to scrub typhus while in the study by Vikrant et al[82] 18.5% of total cases were due to scrub typhus. Much higher number seen in their study is probably due to geographical variation. Our study subjects with scrub typhus-AKI also belonged to hilly area with high scrub typhus incidence.

Renal involvement is a rare manifestation of enteric fever, a water borne diseases of humid climate, occurring only in 2—3% of patients.[111] The spectrum of renal complications in enteric fever includes cystitis, pyelitis, pyelonephritis, mild proteinuria and, less commonly, mild to severe glomerulonephritis, acute tubular necrosis and interstitial nephritis.[112-117] Typhoid glomerulonephritis is a rare complication affecting 2—4% of enteric patients in endemic areas and subjects traveling from endemic areas.[113] Acute renal failure in patients with typhoid fever has been described in several case reports;

dehydration, shock and rhabdomyolysis are the most likely causes.[112] Other rare causes of renal failure include glomerulonephritis, acute tubular necrosis and interstitial nephritis.[112-117] An intrinsic toxic effect of Salmonella on the kidney is suggested by Van Doorn et al.[112] In our study enteric fever accounted for 16% cases (2% of total cases) of tropical AKI in our study. Though enteric is common tropical infection, there is paucity of literature on AKI in enteric fever from india with few studies showing AKI being relatively uncommon in comparison to our study.[118] Our study had relatively more cases probably due to referral bias with sick patients having AKI having higher referral to our centre.

Leptospirosis is another emerging cause of AKI. There is a sharp increase in its incidence during rainy season or after floods. AKI occurs in 20–85% of cases of leptospirosis. Mehta K et al[118] reported 13% incidence of AKI due to leptospirosis. Major factors involved in pathogenesis are direct nephrotoxic action of leptospires, toxin-induced immune response, hyperbilirubinemia, rhabdomyolysis, and hemodynamic alterations due to decrease in peripheral vascular resistance, diarrhea, vomiting, and fever. Basu et al[58] in their study had 3.3% incidence of AKI due to leptospirosis. Kaul et al[72] in their study had 8% incidence of AKI due to leptospirosis. Prakash et al[86] did not note any case of AKI due to leptospirosis during 26 years. In our study leptospirosis was relatively less accounting for 9% of tropical infections and 1.6% of total. Leptospirosis is the leading cause of AKI in Thailand and Singapore. Daher et al[119] in their study of AKI in infectious disease in ICU noted a 11% incidence of leptospiral AKI. Thus, the incidence of AKI due to leptospirosis varies as per the region.

Malarial AKI (MAKI) is considered a disease predominantly of adults and older children,[120-122] specifically in nonimmune adults from areas of relatively low intensity of malaria transmission. Thus, a large number of reports on MAKI have appeared from southeast Asia and the Indian subcontinent where malaria transmission generally is low, with occasional microfoci of intense transmission. The occurrence of AKI in patients from endemic areas with severe malaria is less than 1% to 4.8%, whereas in nonimmune Europeans it is 25% to 30%. The

incidence of AKI in severe P falciparum malaria is as common as cerebral malaria in nonimmune European adults.[123] Almost all patients with fatal cases had AKI and reported a history of recent travel to malaria endemic areas. Only a few reports of MAKI are available from Africa, particularly from areas with intense malaria transmission, where malaria affects mostly young children. In contrast to severe malaria in adults, none of the 180 Gambian children with severe malaria had AKI.[124] However, malaria transmission intensity is not uniform in Africa and a higher incidence of AKI with associated morbidity and mortality has been reported in semi-immune African children.[125] The occurrence of AKI in severe falciparum malaria is quite common in southeast Asia, including India. A significant increase in the incidence of AKI has been reported from several centres across India. In 1982 a study from Orissa indicated cerebral malaria as the predominant presentation of severe malaria. Associated complications of 173 cases of cerebral malaria were AKI (6%) and hepatic failure (1%).[126] Twenty years later, another study from the same area and the same hospital showed 35% of cases with severe P falciparum malaria had AKI. There was a consistent increase in incidence of AKI (from 95 cases in 1994 to 215 cases in 1998).[127] Basu et al[58] reported malaria as the second most common tropical illness of their study accounting for 25.8% of cases of AKI of infectious etiology while Kaul et al[72] noted that malaria was responsible for 18.8% of medical causes of community-acquired AKI. The changes in epidemiology of infections causing community-acquired AKI were highlighted by Prakash et al[86], with a significant increase in the number of AKI cases due to malaria from 4.7% in 1983–1995 to 17.09% in 1996–2008. The majority of the studies had a common inference that the incidence of AKI and jaundice is increasing, and the development of multiple complications result in increased mortality. Studies has also showed increasing incidence of AKI due to malaria with P. vivax.[83,128] In our study malarial AKI was uncommon with 9% of tropical infections and 1.6% of total cases. This was due to geographical variation in incidence of malaria with most cases in

migrant population. They, being from economic weaker section choose a public health centre.

Diarrhoeal diseases causing hypovolemia has been the most common cause of AKI in many older studies from Chennai,[57] Chandigarh (1989),[56] and Boston (1991).[60] Acute diarrheal disease has been decreasing over the past few years due to better hygiene, better facilities, and effective management in primary and secondary level hospitals.[3] While few studies still showed higher rate of AKI due to diarrheal illness (upto 47%)[70,71] in CA-AKI while few studies has shown it to be as low as 6%.[82] Diarrhoeal diseases caused 85% of cases of hypovolemia and 16% of total cases in our study. Higher percentage of cases in our study was probably due in inclusion of CKD population. Persons with CKD and hypertension are the very susceptible to volume loss, possibly due to diminished renal reserve and exacerbating effects of treatment with diuretics and renin-angiotensin-aldosterone system blockers. Proactive management of these unique pharmacologic and physiologic factors is necessary to prevent AKI in this vulnerable population.[129]

Non-infective causes of AKI

Acute pancreatitis accounted for 1.5% of the cases in our study similar to previous study.[82] AKI is one of the most common complications in patients with severe acute pancreatitis. AKI due to severe acute pancreatitis is the result of hypoxemia, the release of pancreatic amylase, impairment of renal microcirculation, decrease in renal perfusion pressure due to abdominal compartment syndrome, or hypovolemia.

Toxin related AKI was observed in ~ 15% of total cases including drugs (9%), poisonings (4%) and snake bite (2%). This data was similar to recent previous studies[79,82] and more in comparison to old literature as suggested by various studies.[56,57,86] In drug induced AKI, NSAIDS were the most common cause, followed by ACEI/ARB and Diuretics, complementary and alternative medications (CAM) and severe hypervitaminosis D. only 25% of drug induced AKI were with regular

prescription, rest all were either non-prescribed or intake of wrong dose or duration of medication.

Though a commonly recognized cause of HA-AKI, it commonly causes CA-AKI especially with complementary and alternative medications and unregulated modern pharmaceutical products.[5]

Most common cause of poisoning was paraquat poisoning with majority of the cases being suicidal followed by accidental. Paraquat is a common herbicidal used in our country mainly due to cheap cost and is still not banned even when its lethality has been clearly documented and there is no antidote. Similar to previous studies, our study had very high mortality (86%) with paraquat mainly due to respiratory failure.[82]

AKI due to snake bite is an important cause of CA-AKI in tropical countries. About 12%– 30% of patients bitten by venomous snakes, primarily vipers, develop AKI. Hemorrhage, hypotension, disseminated intravascular coagulation, intravascular hemolysis, and rhabdomyolysis contribute to the development of AKI. Enzymatic activities of snake venoms account for direct nephrotoxicity. Immunologic mechanism plays a minor role.[5,39-41] We had 2% patients with snake bite-AKI mostly referred from far-flung rural areas even from other states. In previous Indian studies, AKI caused by snakebite was comparatively less which is similar to our study.[72,79] Kaaviya et al[81] reported very high proportion of AKI due to snake bite (49%) probably due to different geographic distribution of snakes as well as population at risk.

Cardiac causes such as myocardial infarction, congestive cardiac failure were the causes of AKI in 4% of our patients, slightly less than what is reported previously.[82] Acute glomerulonephritis was a cause of AKI in 2% of patients as compared to ~ 10% reported in a previous study from India.[57,86]

Surgical – AKI

Surgical causes accounted for AKI in 17.9% patients which is higher than previously reported in other studies.[72,79] It was probably due to inclusion of CKD patients in the study especially secondary to nephrolithiasis. Nephrolithiasis was the most common cause of urinary obstruction similar to previous studies [72,82] either by causing bilateral

obstruction, or bladder outlet obstruction or more commonly unilateral obstruction in patient with solitary functioning kidney. Urinary stone disease in the Indian population is different from that in the Western countries, with a larger percentage of patients having calcium oxalate stones. Previous studies have found calcium oxalate monohydrate as the major component of stones, with hyperoxaluria and hypocitraturia being the most common urinary abnormalities.[130] Dietary factors, inherited metabolic disorders and fluid losses contribute to the increased incidence of nephrolithiasis in the Asian population.[41] Other causes of CA-AKI included BPH, urogenital malignancies, papillary necrosis, urogenital trauma, cystocele and Neurogenic bladder. Urogenital malignancies especially cervical carcinoma if females having urinary obstruction causing AKI is not unheard-of Indian population, and indicate the need for improved screening covering the widest possible population and also regular follow-up of women with pre-cancerous lesions.[131,132]

Some of the earliest case reports of AKI described persons with severe traumatic muscle injuries and subsequent rhabdomyolysis from the battlefield or areas of natural disaster. The classical papers by Bywater and Beall on crush syndrome was indeed the first description of CA-AKI.[25,26] Systemic manifestations that are induced by crush injury are often referred to as crush syndrome. Crush syndrome develops in 30 to 50 percent of cases of traumatic rhabdomyolysis and is frequently seen after catastrophic earthquakes. According to some estimates, the incidence of crush syndrome ranges between 2 and 5 percent of all injured victims of catastrophic earthquakes.[133,136] The incidence of AKI and the frequency with which dialysis is required in these cases have varied widely in different studies. In a report from Bam, Iran, dialysis was required in 6.5 percent of 1975 patients admitted to the hospital.[137] The majority of victims were rescued in less than four hours. In contrast to other reports[138], the authors detected a direct correlation between time under the rubble and outcome. The short time under the rubble might thus explain, at least in part, the lower rate of requiring dialysis in Bam compared with other reports, although this is

not entirely certain. Much higher rates of dialysis requirement were noted in two other catastrophic earthquakes: 54 percent in the Kobe earthquake and 75 percent in the Marmara earthquake.[136,139] In the Kobe earthquake, the need for hemodialysis correlated directly with increased serum creatine kinase (CK) levels as dialysis was required in 84 and 39 percent of patients with a CK level greater or less than 75,000 units/L, respectively.[136] In the Kobe and Marmara earthquakes, the time under the rubble correlated inversely with both serum CK and the frequency of requiring dialysis. A possible explanation for these counterintuitive findings is that victims with more extensive muscle injury and higher CK levels died before they were extricated and transported to the hospital. Although not many other natural disasters cause traumatic rhabdomyolysis, a number of manmade disasters may do so (such as terrorist attacks, mining accidents, wars, and torture). 15 cases in our studies had crush syndrome. They were mainly secondary to high speed road traffic accident mainly affecting age group below 40 years. There is paucity of Indian literature on prevalence and incidence of crush syndrome and AKI. Jayakumar M et al mentioned it to cause 0.6% of total cases.[57]

Obstetric-AKI

AKI is one of the most challenging complications of pregnancy in developing countries and is frequently related to suboptimal antenatal care, out-of-hospital delivery in rural areas, and unsafe abortions conducted by unqualified personnel or using insecure medicines.[5] AKI during pregnancy is uncommon in the developed world. The true incidence is difficult to estimate because of varying diagnostic criteria. Most reviews estimate that, in countries with adequate antenatal care, only approximately 1 in 20,000 pregnancies are affected by AKI severe enough to require renal replacement therapy.[140] Most cases of AKI occur during the third trimester related to preeclampsia, eclampsia, placental abruption, postpartum haemorrhage, disseminated intravascular coagulation, and puerperal sepsis. AKI that is related to pregnancy may also occur in the postpartum period. This may be due to causes listed above that were present prior to delivery that have not

resolved (eg, preeclampsia, HELLP syndrome). Pregnancy-associated atypical HUS usually develops in the postpartum period. AKI secondary to ATN may develop due to hemodynamic stress associated with either hemorrhage or sepsis. Few cases occur early in pregnancy (<20 weeks) is most often due to hyperemesis gravidarum or septic abortion. In addition to these conditions, acute pyelonephritis and, less commonly, urinary tract obstruction have been associated with AKI in pregnant women. The age of the mother is an essential parameter for a multitude of disorders. Several studies implicated a strong association between age and the onset of AKI in pregnancy. According to one report, the occurrence of obstetric AKI in women in the age group of 20-25 years is more frequent.[141] In India, improvements in the antenatal medical care and obstetric care have decreased obstetric AKI to[56,57,76] Medical care for pregnant women is still deficient, and the timing of hospital referral in obstetric AKI is generally delayed. Renal ischemia, caused by haemorrhagic shock or hypotension due to sepsis, is the dominant factor leading to AKI. In our study, obstetric AKI was 6% of total cases mostly in third trimester and post-partum with mean age of 28 years. 48% were due to sepsis, 26% were a part of preeclampsia, 17% due to Hemorrhage and rest were due to HELLP and HUS. Results were similar to studies done in recent past.[72,82]

OUTCOME VARIABLES

Need of ICU admission

In our present study, a total of 159 patients (41.8%) patients required ICU stay with average ICU stay of 6.8 day. Only 13% patient of CAAKI in study by goswami et al[71] and only 6% in study by vikrant et al[82] had need of ICU. Much more patients requiring ICU admission in our centre could be related to more severe or more delayed presentation as most of these were on assisted ventilation. On comparing various age groups, there was no significant difference in the need of ICU admission on univariate analysis. This finding is probably because of more severity of underlying illness like crush injury, poisonings, and snake bite in younger population. Gender also had no significant difference in the need of ICU admission on univariate analysis. Those

coming from rural area had relatively more severity of illness and had significantly higher need of ICU admission on univariate analysis. This probably reflects delay in diagnosis and treatment of AKI more in rural population and illness compounded by indigenous treatment. Though this was statistically insignificant in multivariate analysis with p-value 0.07, its clinical significance cannot be undermined. On univariate analysis, diabetics, those with underlying CKD, CAD and CLD had significantly higher chance of need of ICU admission, while on multivariate analysis CKD as well as CLD patients had statistically significant higher need of ICU admission. Also, more the number of comorbidities, more the patients had need of ICU in univariate analysis but not in multivariate analysis. This was probably because most of these patients with multiple comorbidities had sepsis-AKI, an independent parameter in both univariate as well multivariate analysis. Apart from sepsis, patients with poisoning as well as crush syndrome had statistically more need of ICU admission on multivariate analysis. Patient with tropical fever and cardiorenal syndrome had significantly higher ICU admission on univariate analysis but on multivariate analysis. Patients with hypovolemia, drug induced AKI and urinary obstruction had significantly less need of ICU admission. On comparing medical vs surgical vs obstetric cases, no significant results were obtained. Severity of AKI had significant correlation with need of ICU on univariate but was probably a confounder as on multivariate, it was insignificant. Oliguric patients, patients requiring inotropic support and patients requiring dialysis had significantly higher ICU admission on univariate analysis but on multivariate only patients with inotropic support had significant association with need of ICU. Few patients were on inotropic support outside ICU, mainly in the step-down HDU units. Modes of dialysis other than HD had significantly higher ICU admission on univariate analysis but was probably a confounder of hypotension and overall sickness as these modalities were only given to hemodynamically unstable patients. On multivariate, various modes of HD had no significance. Patients who had complete recovery had significantly low ICU admission rate well correlating their relatively

stable hemodynamic and better outcome. Patients who had partial or no recovery had insignificant results. In most previous studies, this aspect is either studied mainly for HA-AKI or not analysed. In a previous study by Abd El Hafeez S[142], In multivariable analysis, previous use of diuretics, previous use of angiotensin receptor blockers, history of sepsis, and level of education were associated with need of ICU stay. Common thing was sepsis being significantly associated with Need of ICU in both the studies. Some factors might have given insignificant results due to lack of adequate power of study.

Need of assisted ventilation

In our present study, a total of 115 patients (30%) patients required assisted ventilation. In study by goswami et al[71] only 7% patients of CAAKI required assisted ventilation. Much more patients requiring assisted ventilation in our centre could be related to more severe or more delayed presentation. On comparing various age groups, there was no significant difference in the need of assisted ventilation on univariate analysis. Gender also had no significant difference in the need of assisted ventilation on univariate analysis. Those coming from rural area also had no significant difference in the need of assisted ventilation than urban population on univariate analysis. On univariate analysis, diabetics, those with underlying CKD, and COAD had significantly higher chance of need of assisted ventilation, while on multivariate analysis only diabetics had statistically significant higher need of assisted ventilation. Also, more the number of comorbidities, more the patients had need of assisted ventilation in univariate analysis as well as multivariate analysis. Apart from sepsis, patients with poisoning as well as crush syndrome had statistically more need of assisted ventilation on multivariate analysis. Patient with cardiorenal syndrome had significantly higher assisted ventilation on univariate analysis but on multivariate analysis. Patients with hypovolemia, drug induced AKI and urinary obstruction had significantly less need of assisted ventilation. On comparing medical vs surgical vs obstetric cases, no significant results were obtained. Severity of AKI had significant correlation with need of assisted ventilation on univariate but

was probably a confounder as on multivariate, it was insignificant. Oliguric patients, patients requiring inotropic support and patients requiring dialysis had significantly higher assisted ventilation on univariate analysis but on multivariate only patients with inotropic support had significant association with need of assisted ventilation. Modes of dialysis other than HD had significantly higher assisted ventilation on univariate analysis but on multivariate, various modes of dialysis had no significance in relation to need of assisted ventilation. Patients who had complete recovery had significantly low assisted ventilation need while patients who had partial or no recovery had insignificant results. In most previous studies, this aspect is either studied mainly for HA-AKI or not analysed. Some factors might have given insignificant results due to lack of adequate power of study. The factors affecting need of assisted ventilation were quite similar, as expected; to those with need of ICU admission as 72% patients of ICU required assisted ventilation.

Need of Renal replacement therapy

In our present study, a total of 123 patients (32.3%) patients required dialysis. In study by Anupama kaul et al[72] 83% patient required dialysis probably because it was a study based of nephrology unit only. Other studies like Goswami et al[71] and vikrant et al reported need of dialysis in 30% and 23% respectively comparable to our study. Mostly traditional indications of refractory hyperkalaemia, metabolic acidosis, fluid overload or features of uraemia were used to guide need of dialysis. Most common dialytic modality used was hemodialysis used in 50 cases (40.7%). It was used in hemodynamically stable patients with 52% patients not requiring ICU care. Rest all modality of dialysis were performed in ICU setting in hemodynamically unstable patients, most commonly Sustained low efficiency dialysis (SLED) in 37 patients followed by peritoneal dialysis in 28 patients (PD) and CRRT in 8 patients. On comparing various age groups, numerically more percentage of elderly required dialysis but on univariate analysis there was no significant difference in the need of dialysis. This finding is probably because of more severity of underlying illness like crush

injury, poisonings, and snake bite in younger population. Gender also had no significant difference in the need of dialysis on univariate analysis. Those coming from rural area had no significant difference in the need of dialysis from urban dweller on univariate analysis. On univariate analysis, diabetics, hypertensives, those with underlying CKD, and CLD had significantly higher chance of need of dialysis, while on multivariate analysis only underlying CKD had statistically significant higher need of dialysis. Also, more the number of comorbidities, more the patients had need of dialysis in univariate analysis as well as multivariate analysis. Sepsis was an independent parameter in both univariate as well multivariate analysis for need of dialysis. Apart from sepsis, patients with snake bite had statistically more need of dialysis admission on multivariate analysis. Patient with poisoning had significantly higher dialysis on univariate analysis but not on multivariate analysis. Patients with hypovolemia, and urinary obstruction had significantly less need of dialysis. On comparing medical vs surgical vs obstetric cases, no significant results were obtained. Patients requiring inotropic support, ICU and assisted ventilation had significantly higher dialysis on univariate analysis but on multivariate only patients with inotropic support had significant association with need of dialysis. Patients who had complete recovery had significantly low dialysis need well correlating with their relatively milder AKI and better outcome. Patients who had partial recovery had insignificant results while patients with non-recovery had significant more need of dialysis as expected. In the study by Anupama kaul et al[72] medical, surgical as well as obstetric patients had no difference in the need of dialysis which was up to the tune of ~80%. Similarly, various etiologies had no significant difference in need of dialysis. Their most common modality was also hemodialysis. Literature on the choice and time of initiation of dialytic modality is conflicting and no study has conclusively demonstrated the appropriate type and appropriate dose of RRT in AKI.[9,18,19,20,21,72]

Length of stay

Average hospital stays for CA-AKI patients were 9.4 days in our study with non-survivors having 8.8 days average stay. On comparing various age groups, people with age below 30 had statistically more prolonged hospital stay than other age groups on univariate analysis but not on multivariate analysis. This finding is probably because of more number of illnesses with prolonged stay like crush injury, poisonings, and snake bite in younger population. Gender had no significant difference in the length of hospital stay on univariate analysis. Those coming from rural area had no significant difference in the length of hospital stay from urban dweller on univariate analysis. On univariate analysis, none of the comorbidities had significant difference in the length of hospital stay. Even patients without comorbidities had overall similar duration of stay as they comprise patients who had etiology like hypovolemia (much shorter stay) as well as crush syndrome, poisonings and snake bite (much longer stay) with average having no difference than those with patient with comorbidities. Also increasing number of comorbidities had no effect of duration of hospital stay. Patient with poisoning, snake bite and crush syndrome, all had significantly longer hospital stay both on univariate analysis as well as on multivariate analysis with a p-value of 0.03, 0.02 and 0.02 respectively. Patients with hypovolemia had significantly shorter hospital stay. On comparing medical vs surgical vs obstetric cases, surgical patients had significantly longer stay on univariate analysis but not on multivariate (p value – 0.34). It was probably because crush injury patients comprise significant number of surgical patients hence creating a confounder eliminated on multivariate analysis. Patients with stage 1 and 2 of KDIGO - AKI staging had significant shorter stay than stage 3 on univariate analysis but this factor also was eliminated on multivariate analysis. Patients requiring inotropic support, assisted ventilation and dialysis had significantly longer hospital stay on univariate analysis as well as multivariate analysis. Patient requiring ICU admission and oliguric patients had significantly longer stay on univariate analysis but not on multivariate

analysis. Comparing different modalities of dialysis, HD patients had longer stay on univariate analysis but not on multivariate analysis. This finding may be because HD patients were surviving and non-recovering because patients with non-recovery at 1 month had significantly prolonged stay on both univariate as well multivariate analysis. Patients with partial recovery had prolonged stay on univariate analysis but not on multivariate analysis. Patients with complete recovery had shorter stay on univariate analysis but not on multivariate analysis. Study by Anupama kaul et al on CA-AKI showed an average hospital stay of 43- 49 days.[72] Most other studies discuss this parameter for HA-AKI with duration of stay ranging from 10-25 days.[68] Our study report of ~ 10 days of average hospital stay is probably due to economic bias with patients being shifted to a step-down health facility early.

SHORT TERM MORTALITY

7 day mortality

Early mortality (within 7 days) in our study was high (11%). 75% cases of early mortality were due to sepsis in patients with underlying multiple comorbidities mostly diabetes, hypertension, CKD and CLD. 6 cases due to tropical fever mainly dengue, 2 cases of HRS and one case each of drug induced AKI and hypovolemia. Though higher proportion of elderly had early mortality, it was statistically not significant. Gender as well as residential status had no effect on early mortality. Most of the mortality was in stage 3 AKI and that was significant on univariate but not on multivariate analysis. Oliguria, need of ICU, dialysis, mode of dialysis, inotropes, and assisted ventilation, all had significant association with early mortality on univariate analysis but on multivariate, only need of dialysis and need of inotropes had significant association. Over all on multivariate analysis, higher number of comorbidities, Sepsis, need of dialysis and need of inotropic support were significantly associated with early mortality. Previous studies have discussed early mortality in context of HA-AKI or sepsis-AKI. Our result is in concordance with previous studies showing high mortality in Sepsis- AKI especially in patients with burden of comorbidities.[143]

Sepsis being common cause of early mortality is not surprising, as the pathophysiology of AKI in sepsis is complex and includes pre-renal factors due to volume depletion, systemic vasodilatation, and septic shock, leading to hypoperfusion and ischemic acute tubular necrosis. Infections result in the release of endotoxins and widespread damage; this can predispose the patient to multiorgan failure, which carries a grave prognosis.

30 day mortality

Similar to early mortality age, gender and residential status had no effect on overall mortality; Sepsis was the most common cause of overall 30-day mortality as well. Overall 34% patient with sepsis-CA-AKI succumbed to their illness. On univariate analysis 30-day mortality was significantly associated with diabetes, hypertension, underlying CKD, CVA and CLD. People with more number of comorbidities had high mortality. People with sepsis and poisoning had significantly higher mortality. Increasing stage of AKI, oliguria, need of ICU, dialysis, Ventilation, inotropes, dialysis modality other than HD, all were significantly associated with higher mortality. This suggests that those patients who were very sick had significantly high mortality. Though there was difference between the survivors and non-survivor's with Medical AKI having higher percentage of mortality, but it was not statistically significant with a p-value of 0.20.

Survival analysis showed that most of the mortality in obstetric group was early mortality while other groups had both early as well as late mortalities. Patients with tropical etiologies had higher mortality in age below 40 year on univariate analysis. This was probably due to etiologies like poisoning, snake bite and crush injury commoner in this age group. Patients with non-tropical etiologies mainly sepsis, as expected, had higher mortality in age above 40 years. On multivariate analysis, underlying CKD and CLD, higher number of comorbidities, Sepsis, poisoning, need of dialysis and need of inotropic support were significantly associated with mortality. In the study by Anupama kaul et al[72], overall in-hospital mortality among patients with CAAKI was 26.2% with sepsis having highest in-hospital mortality. Similar to our

study, need for assisted ventilation, hypotension, oliguria and coexisting liver disease were associated with increased in-hospital mortality. Mortality rate in study by Vikrant et al[82] was 8.7%, much lower than our study. Presence of anaemia, use vasopressor drugs, and requirement of ICU support were independent predictive factors for mortality in their study. In study by Kohli et al[78] also, sepsis as cause of ARF and critical illness were independent predictor of mortality. Neither previously existing CAD nor cardiorenal AKI was associated with increased mortality in our study though they were associated with higher partial recovery. It is difficult to explain, may be by chance or may be AKI in these patients is permissive-AKI with better cardiac outcome.[144]

Complete, Partial and Non recovery.

45% of our patients out of total cases achieved complete renal recovery, 27% had partial recovery while 9% had no renal recovery with 5 % remaining dialysis dependent. Patients who recovered completely were younger in age, had no or single comorbidity mostly hypertension had much less need of ICU admission, assisted ventilation, need of dialysis, a shorted hospital stay in comparison to patients with partial or non-recovery. Patients with Diabetes, underlying CKD, CAD and COAD had less chance of complete recovery from the current CA-AKI episode on univariate analysis. Patients with etiologies like Cardiorenal syndrome, drug induced AKI, sepsis, snake bite, tropical fever and multifactorial AKI had less chance of complete renal recovery in comparison to patients without these etiologies. On contrary, patients with Hypovolemia had better chances of renal recovery. CA-AKI Patients requiring ICU stay, inotropic support, mechanical ventilation, dialysis, patients who had multiorgan failure, oliguria had significantly lesser chances of renal recovery in comparison to those without it suggesting that severity of underlying comorbidities, etiology and CA-AKI had a significant relation to outcome. In study by kohli et al[78] 40.1% had complete renal recovery, 3.7% patients remained dialysis-dependent, and 8.5% made an incomplete renal recovery. Our results were quite similar with more partial recovery and lesser morality. In

study of kaaviya et al also, 45% had complete renal recovery, 47.4% had partial renal recovery, whereas 7.6% did not have renal recovery.

Our study has certain limitations. First, because the study was carried out at tertiary care hospital, it does not truly reflect the etiological spectrum of AKI prevalent in the community. Many patients don't take regular medical consultation with illness and follow indigenous or home remedies hence cannot be accounted for in hospital-based study. Also, milder AKI may go unnoticed in the community which may have long term consequences but is not captured in hospital-based analysis. Second, pediatric patients were excluded. Finally, long-term outcome could not be studied in our patients as follow up were limited to one-month post discharge only. Nevertheless, our study describes very relevant information on adult AKI spectrum encountered at a large tertiary care referral centre in a developing country. Our study included patients with underlying CKD and incident CKD was also included, which are mostly excluded in other studies.

SUMMARY AND CONCLUSION

- The study was done to understand the epidemiology and outcome of CA-AKI in our region. Incidence of CAAKI has increased with time probably due to better identification, documentation and changing definitions
- We prospectively studied 380 patients after screening 54285 patients with overall 7 patients per 1000 admission having CA-AKI.
- Mean age of AKI is still much lower than the mean age in western population, even for etiologies not limited to tropical climate like sepsis. CAAKI in the middle-aged population creates a huge economic burden on families.
- Though statistically not significant, rural population had more need of ICU stay, had longer hospital stay and had higher rate of non or partial recovery. It gives us an indirect estimate of delay in diagnosis and treatment of AKI and their illness compounded by indigenous treatment.
- Our study subjects seem to arise from 2 different populations, one population relatively young with no or single comorbidity at risk of tropical etiologies while another population, relatively old with comorbidities complicated by non-tropical etiologies. This is probably due to social inequalities in our population, those with limited resources still affected by common tropical etiologies.
- Our study group had high rate of comorbidities mainly in age group above 50. With increasing age number of comorbitidies increases. This population may benefit from more frequent blood tests in the event of an acute illness or medication change to screen early AKI and early institution of care.
- With many patients with pre-renal AKI (hypoperfusion at onset) progressing to renal and very few cases of stage 1 AKI, health education at primary level is needed for early treatment of dehydration. Also, crucial time wasted in alternative treatment needs to be highlighted.

- Sepsis is now the commonest cause of AKI both CA-AKI and HA-AKI mainly in medical patients but also in surgical patients. Though obstetric AKI is relatively less common in comparison to previous era, still puerperal sepsis is most common cause of obstetric AKI. Increase in Sepsis-AKI also actually highlight the fall in number of cases of tropical etiologies. Most common source of sepsis is the lungs followed by pyelonephritis.
- Dengue and scrub typhus are common causes of Tropical-AKI in this geographical region, requiring preventive measures as well as early detection and treatment.
- Diarrhoeal diseases causing hypovolemia has been the most common cause of AKI in the past. Decreasing incidence over the past few years has been documented which is due to better hygiene, better facilities, and effective management in primary and secondary level hospitals.
- Persons with CKD and hypertension are the very susceptible to volume loss, possibly due to diminished renal reserve and exacerbating effects of treatment with diuretics and renin-angiotensin-aldosterone system blockers. Proactive management of these unique pharmacologic and physiologic factors is necessary to prevent AKI in this vulnerable population
- Though a commonly recognized cause of HA-AKI, Drug induced AKI commonly causes CA-AKI especially with complementary and alternative medications and unregulated modern pharmaceutical products. NSAIDS were the most commonly known cause, followed by ACEI/ARB and Diuretics. Only 25% of drug induced AKI were with regular prescription, rest all were either non-prescribed or intake of wrong dose or duration of medication. Severe hypervitaminosis D as a cause of AKI also needs attention causing unnecessary financial burden on society.
- Paraquat poisoning is the most common poisoning observed in this geographical area causing AKI. It is a common herbicidal, still commonly use in India with no proper safety measures, its mostly

lethal and there is no antidote. A ban on this chemical is highly recommended.

- Snake bite is an important cause of CA-AKI in our country. Different geographic distribution of snakes as well as population at risk determines AKI incidence. Our study had much smaller number of cases mostly from far-flung area.
- Nephrolithiasis was the most common cause of urinary obstruction causing AKI either by bilateral obstruction, bladder outlet obstruction or more commonly unilateral obstruction in patient with solitary functioning kidney.
- High speed road traffic accident mainly affecting young population causing crush syndrome/ rhabdomyolysis was an important cause of AKI in surgical patients.
- Improvements in the antenatal medical care and obstetric care have decreased obstetric AKI, still medical care for pregnant women is deficient, and the timing of hospital referral many a times is delayed, leading to AKI, mostly in third trimester and post-partum. Most common cause of AKI was sepsis, followed by preeclampsia and Hemorrhage.
- Patients with underlying diabetes, CKD or CLD, patients with higher number of comorbidities, patients having AKI due to Sepsis, poisoning, crush injury and patients requiring inotropic support requires ICU admission and assisted ventilation more frequently.
- Patients with underlying CKD, higher number of comorbidities, patients having AKI due to Sepsis and snake bite, and patients requiring inotropic support requires dialysis more frequently.
- Patients with poisoning, crush injury, snake bite, patients requiring inotropes, ventilation, and dialysis had significantly longer hospital stay.
- Patients with higher number of comorbidities, AKI due to Sepsis, patients requiring dialysis and inotropic support had much higher early mortality (7-day mortality). Overall mortality (30-day mortality) was affected by underlying CKD and CLD, higher

number of comorbidities, Sepsis, poisoning, need of dialysis and inotropic support.

- 45% of our patients out of total cases achieved complete renal recovery, 27% had partial recovery. 9% patients had no renal recovery and 5 % remained dialysis dependent. Patients who recovered completely were younger in age, had no or single comorbidity mostly hypertension had much less need of ICU admission, assisted ventilation, need of dialysis, a shorted hospital stay in comparison to patients with partial or non-recovery.

Under the light of above-mentioned results, following conclusion was withdrawn:

- The clinical and epidemiological characters of CAAKI have changed over the years. Management of AKI has also advanced during this period. Still, unmet needs in the area of CA-AKI are high. Sepsis has emerged as the most common cause as well as killer in CA-AKI similar to HA-AKI, still preventable causes of AKI like gastroenteritis, tropical infections are common requiring better public health initiatives. Relatively old patients having comorbidities complicated by non-tropical etiologies mainly sepsis and drugs and have increased. Poisoning, crush injuries and snake bite causes high morbidity and mortality is young population. Patients with multiple comorbidities having severe AKI stage, having oliguria, requiring ICU, requiring ventilation, requiring inotropes, requiring dialysis modality other than HD i.e overall sick patients had much high mortality. With 19% mortality and only 45% complete recovery, it highlights need of better preventive strategies. We recommend that additional efforts be made especially by epidemiologists to rectify things at grass-root level. Efforts like better implementation of vector disease program, awareness camps for timely rehydration, timely referral, better regulation of drugs mainly painkillers, banning paraquat and similar lethal poisons, further improving maternal health care, early screening of high risk population, measures to control the

silent epidemic of non-communicable disease and awareness that alternative medicines aren't free from ill-effects rather causes delay in diagnosis and treatment. Regarding sepsis, the need of hour is antibiotic stewardship to avoid antibiotic resistance at community level. Multidrug resistant pathogens in community acquired sepsis will be like a ticking time bomb. We also recommend studies at community level and with longer follow-up for better understanding of epidemiology, natural course and outcome of CA-AKI. We also feel the need of better biomarkers and biomarker based AKI severity for early detection and management of AKI.

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S.no.	Name	MRD No.	Age	Gender	Rural/ Urban	Comorbidities	Comorbidity COAD	Comorbidity CLD	Comorbidity DM	Comorbidity HTN	Comorbidity CKD known	Comorbidity CKD (total)	Comorbidity CAD	Comorbidity HCV	Comorbidity HBV	Comorbidity HIV	Comorbidity MALIGNANCY	Comorbidity NEPHROLITHIASIS	Comorbidity TB	Comorbidity RA	Comorbidity CVA	Primary Etiology OF AKI	MULTIFACTORIAL AKI	ETIOLOGY GROUP
1	S.K.	445657	48	F	R	2	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	DRUG- NSAIDS	YES	DRUG
2	S.S.	207420	65	F	R	≥ 3	NO	YES	YES	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	YES	LVF/CARDIORENAL	YES	CARDIORENAL
3	K.K.	448416	69	F	R	> 3	NO	NO	YES	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	YES	CARDIORENAL
4	A.C.	437624	50	M	R	> 3	YES	NO	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	SEPSIS/PNEUMONIA	NO	SEPSIS
5	H.S.	447603	68	M	U	2	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-SEPSIS/D.FOOT	NO	SEPSIS
6	A.M.	446513	54	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/URETHRAL INJURY	NO	U.O.
7	B.S.	414356	65	M	U	2	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	SUR-BPH	NO	U.O.
8	U.B.	386684	42	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
9	K.K.G.	445196	44	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
10	B.K.	445910	51	M	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	BURKIT/ TLS	NO	TLS
11	B.K.	436839	33	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
12	T.S.	447476	62	M	U	2	NO	NO	YES	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	NO	DRUG
13	K.R.	447531	50	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-CYSTOCELE	NO	U.O.
14	G.B.	447613	53	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
15	S.K.	447567	31	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PPH	NO	HYPOVOLEMIA
16	B.S.	449261	43	M	U	2	NO	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
17	K.S.	448370	64	M	U	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	SUR-Ca PROSTATE	NO	U.O.
18	D.S.	447444	33	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-PERFORATION PERITONITIS	NO	SEPSIS
19	S.S.	446563	67	F	U	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	YES	SEPSIS
20	R.S.	445158	48	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
21	A.B.	447608	62	M	R	2	NO	NO	YES	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	SUR-PAPILLARY NECROSIS	NO	U.O.
22	S.K.G.	209943	40	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
23	M.S.	440867	18	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	G6PD/HEMOLYSIS/AKI	NO	G6PD
24	M.B.	448428	49	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/SEPTIC ARTHRITIS	NO	SEPSIS
25	M.B.S.	447628	51	M	U	≥ 3	YES	YES	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO	DRUGS-RIFAMPICIN	NO	DRUG
26	J.S.	446621	23	M	U	≥ 3	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
27	S.K.	178730	53	F	R	2	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
28	R.L.M.	271565	52	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/INFECTIVE ENDOCARDITIS	NO	SEPSIS
29	G.S.	143762	19	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	GLOMERULONEPHRITIS	NO	GLOMERULONEPHRITIS
30	S.L.S.	448427	51	M	U	2	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	HRS	NO	HRS
31	V.S.	448499	43	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- ARB	NO	DRUG
32	R.G.	449169	36	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
33	D.K.	436859	54	F	U	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
34	D.S.	302178	65	M	U	0	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-BPH	NO	U.O.
35	S.S.	423442	49	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-SEPSIS/BURNS	NO	SEPSIS
36	S.K.	441759	69	M	U	2	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-Ca PROSTATE	NO	U.O.
37	D.P.A.	433561	54	M	U	1	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
38	S.S.	446240	46	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
39	S.S.	186022	38	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
40	B.K.	34433	48	F	U	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
41	G.S.	187973	46	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-MALARIA	NO	TROPICAL FEVER
42	M.S.	446812	51	M	R	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
43	A.S.	441940	50	M	U	2	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	A.G.E	NO	HYPOVOLEMIA
44	G.R.	263742	40	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
45	S.S.	431887	39	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OPC POISONING	NO	POISONING
46	S.S.	445212	43	M	U	≥ 3	NO	YES	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
47	M.D.	440477	26	F	R	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	YES	DRUG
48	G.S.	413875	41	M	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
49	K.R.	447509	58	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
50	I.S.	382618	59	M	R	1	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
51	A.K.	449037	52	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
52	S.P.	442044	60	M	U	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-BPH	NO	U.O.
53	R.M.	73842	19	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	PARAQUOT POISONING	NO	POISONING
54	A.S.	403322	48	M	U	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- ARB	YES	DRUG
55	V.R.	448467	42	F	R	2	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	YES	SEPSIS
56	G.K.	443654	29	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREPUREAL SEPSIS	NO	SEPSIS
57	G.S.	449429	57	M	U	≥ 3	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
58	R.A.	192383	53	M	R	≥ 3	NO	YES	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	ACUTE PANCREATITIS	NO	HYPOVOLEMIA
59	P.K.	414875	41	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
60	K.D.	449712	42	F	R	1	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
61	M.D.	382985	44	F	R	≥ 3	YES	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
62	S.K.	449836	52	M	R	2	NO	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
63	I.S.	438417	50	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/GASTROENTERITIS	NO	SEPSIS
64	B.S.	447504	33	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-PERFORATION PERITONITIS	NO	SEPSIS
65	B.D.	428483	27	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PPH	NO	HYPOVOLEMIA
66	N.J.K.	193851	50	F	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	SUR-Ca CERVIX	NO	U.O.
67	A.S.	449931	45	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
68	M.R.	449952	51	M	R	2	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-SEPSIS/D.FOOT	NO	SEPSIS
69	V.D.	430907	21	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREECLAMPSIA	NO	PREECLAMPSIA
70	S.S.	449953	22	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
71	K.L.	448313	53	M	U	≥ 3	NO	NO	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	YES	DRUG
72	S.C.B.	449979	29	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
73	K.L.	450006	53	F	R	2	NO	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	SUR-CYSTOCELE	NO	U.O.
74	S.D.	449273	41	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
75	L.S.	166568	43	F	R	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
76	V.S.	450020	41	M	U	≥ 3	NO	NO	NO	NO	NO	YES	NO	YES	YES	YES	NO	YES	YES	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.

S.no.	Name	MRD No.	Medical AKI	Surgical AKI	Obstetric AKI	TYPE OF AKI	stage	Initial dialysis modality	Hospital stay	ICU Stay	HYPOTENSION	OLIGURIA	outcome discharge	Outcome 1 month	Readmission	Biopsy	Infective	Tropical etiology	Need of inotropes
1	S.K.	445657	YES	NO	NO	renal	3	ND	8	0	NO	NO	NR	NR	YES	NO	no	yes	NO
2	S.S.	207420	YES	NO	NO	prerenal	3	SLED	13	6	YES	YES	PR	PR	YES	NO	no	no	YES
3	K.K.	448416	YES	NO	NO	prerenal	3	SLED	8	6	YES	YES	PR	DEATH,19	YES	NO	no	no	YES
4	A.C.	437624	YES	NO	NO	renal	3	SLED	6	6	YES	NO	DEATH,6	DEATH,6	NA	NO	yes	no	YES
5	H.S.	447603	NO	YES	NO	renal	3	HD	10	0	YES	YES	PR	PR	YES	NO	yes	no	YES
6	A.M.	446513	NO	YES	NO	postrenal	3	HD	10	0	NO	NO	PR	PR	NO	NO	no	no	NO
7	B.S.	414356	NO	YES	NO	postrenal	3	ND	11	0	NO	NO	PR	PR	YES	NO	no	no	NO
8	U.B.	386684	YES	NO	NO	prerenal	1	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
9	K.K.G.	445196	YES	NO	NO	prerenal	2	ND	6	0	YES	NO	CR	CR	NO	NO	no	yes	NO
10	B.K.	445910	YES	NO	NO	renal	3	HD	13	0	NO	YES	PR	PR	YES	NO	no	no	NO
11	B.K.	436839	NO	YES	NO	postrenal	3	ND	10	0	NO	NO	PR	PR	NO	NO	no	yes	NO
12	T.S.	447476	YES	NO	NO	renal	3	ND	12	0	NO	NO	NR	NR	YES	NO	no	yes	NO
13	K.R.	447531	NO	YES	NO	postrenal	2	ND	6	0	NO	NO	PR	CR	NO	NO	no	no	NO
14	G.B.	447613	YES	NO	NO	renal	2	ND	8	0	YES	NO	PR	CR	NO	NO	yes	no	YES
15	S.K.	447567	NO	NO	YES	prerenal	3	ND	7	2	YES	NO	PR	CR	NO	NO	no	yes	NO
16	B.S.	449261	YES	NO	NO	prerenal	2	ND	6	0	YES	NO	CR	CR	NO	NO	no	yes	NO
17	K.S.	448370	NO	YES	NO	postrenal	3	HD	8	0	NO	NO	PR	PR	YES	NO	no	no	NO
18	D.S.	447444	NO	YES	NO	prerenal	3	CRRT	22	17	YES	YES	PR	PR	YES	NO	yes	no	YES
19	S.S.	446563	YES	NO	NO	renal	2	ND	7	0	NO	NO	PR	PR	NO	NO	yes	no	NO
20	R.S.	445158	YES	NO	NO	prerenal	1	ND	5	0	YES	YES	PR	CR	NO	NO	no	yes	NO
21	A.B.	447608	NO	YES	NO	postrenal	3	ND	14	0	NO	NO	PR	PR	YES	NO	no	no	NO
22	S.K.G.	209943	YES	NO	NO	prerenal	2	ND	5	0	YES	NO	PR	CR	NO	NO	no	yes	NO
23	M.S.	440867	YES	NO	NO	renal	3	ND	16	0	NO	NO	PR	CR	NO	NO	no	yes	NO
24	M.B.	448428	YES	NO	NO	renal	3	ND	11	0	YES	NO	PR	PR	NO	NO	yes	no	YES
25	M.B.S.	447628	YES	NO	NO	renal	3	SLED	6	6	YES	NO	DEATH,6	DEATH,6	NA	NO	no	yes	YES
26	J.S.	446621	NO	YES	NO	postrenal	2	ND	8	0	NO	NO	PR	CR	NO	NO	no	yes	NO
27	S.K.	178730	NO	YES	NO	postrenal	3	ND	17	6	NO	NO	PR	PR	YES	NO	no	yes	NO
28	R.L.M.	271565	YES	NO	NO	renal	3	ND	10	0	YES	NO	NR	CR	YES	NO	yes	no	YES
29	G.S.	143762	YES	NO	NO	renal	3	ND	10	0	NO	NO	PR	CR	NO	AGN- IgA NEPH	no	no	NO
30	S.L.S.	448427	YES	NO	NO	prerenal	3	CRRT	4	4	YES	YES	DEATH,4	DEATH,4	NA	NO	no	no	YES
31	V.S.	448499	YES	NO	NO	renal	1	ND	3	0	NO	NO	CR	CR	NO	NO	no	no	NO
32	R.G.	449169	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
33	D.K.	436859	YES	NO	NO	renal	3	ND	7	0	YES	NO	NR	NR	YES	NO	yes	no	YES
34	D.S.	302178	NO	YES	NO	postrenal	3	ND	6	0	NO	NO	PR	PR	NO	NO	no	no	NO
35	S.S.	423442	NO	YES	NO	prerenal	2	ND	6	0	NO	NO	PR	CR	NO	NO	yes	no	NO
36	S.K.	441759	NO	YES	NO	postrenal	3	ND	6	0	NO	NO	PR	PR	YES	NO	no	no	NO
37	D.P.A.	433561	YES	NO	NO	renal	3	ND	9	0	YES	NO	NR	CR	YES	NO	yes	no	YES
38	S.S.	446240	YES	NO	NO	prerenal	1	ND	5	0	YES	NO	CR	CR	NO	NO	no	yes	NO
39	S.S.	186022	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
40	B.K.	34433	YES	NO	NO	renal	3	ND	9	0	YES	NO	PR	CR	YES	NO	yes	no	YES
41	G.S.	187973	YES	NO	NO	renal	3	SLED	12	12	YES	YES	DEATH,12	DEATH,12	NA	NO	yes	yes	YES
42	M.S.	446812	YES	NO	NO	renal	3	PD	13	10	YES	YES	PR	PR	YES	NO	yes	no	YES
43	A.S.	441940	YES	NO	NO	prerenal	2	ND	5	0	YES	NO	PR	CR	NO	NO	no	yes	NO
44	G.R.	263742	YES	NO	NO	prerenal	1	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
45	S.S.	431887	YES	NO	NO	prerenal	3	ND	17	10	YES	NO	PR	CR	NO	NO	no	yes	YES
46	S.S.	445212	YES	NO	NO	renal	3	SLED	5	5	YES	NO	DEATH,5	DEATH,5	NA	NO	yes	no	YES
47	M.D.	440477	YES	NO	NO	renal	3	ND	12	0	NO	NO	NR	NR	YES	AIN	no	yes	NO
48	G.S.	413875	NO	YES	NO	postrenal	2	ND	7	0	NO	NO	CR	CR	NO	NO	no	yes	NO
49	K.R.	447509	NO	YES	NO	renal	3	ND	16	11	YES	NO	PR	CR	NO	NO	no	no	NO
50	I.S.	382618	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	PR	CR	NO	NO	no	yes	NO
51	A.K.	449037	YES	NO	NO	renal	2	ND	6	0	NO	NO	PR	CR	NO	NO	yes	no	NO
52	S.P.	442044	NO	YES	NO	postrenal	3	ND	5	0	NO	NO	PR	CR	YES	NO	no	no	NO
53	R.M.	73842	YES	NO	NO	renal	3	ND	23	23	NO	NO	DEATH,23	DEATH,23	NA	NO	no	yes	NO
54	A.S.	403322	YES	NO	NO	renal	2	ND	4	0	NO	NO	CR	CR	NO	NO	no	no	NO
55	V.R.	448467	YES	NO	NO	renal	3	PD	3	3	YES	YES	DEATH,3	DEATH,3	NA	NO	yes	no	YES
56	G.K.	443654	NO	NO	YES	renal	3	ND	9	0	YES	NO	PR	PR	NO	NO	yes	yes	YES
57	G.S.	449429	YES	NO	NO	renal	3	SLED	7	7	YES	NO	DEATH,7	DEATH,7	NA	NO	yes	no	YES
58	R.A.	192383	YES	NO	NO	prerenal	3	SLED	19	11	YES	YES	PR	PR	YES	NO	no	no	NO
59	P.K.	414875	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
60	K.D.	449712	NO	YES	NO	postrenal	3	HD	6	0	NO	NO	PR	PR	NO	NO	no	yes	NO
61	M.D.	382985	YES	NO	NO	renal	3	SLED	3	3	YES	YES	DEATH,3	DEATH,3	NA	NO	yes	no	YES
62	S.K.	449836	YES	NO	NO	renal	2	ND	11	0	NO	NO	NR	PR	NO	NO	yes	no	NO
63	I.S.	438417	YES	NO	NO	prerenal	3	ND	10	0	YES	NO	PR	PR	NO	NO	yes	no	YES
64	B.S.	447504	NO	YES	NO	prerenal	3	ND	14	9	YES	NO	PR	CR	NO	NO	yes	no	YES
65	B.D.	428483	NO	NO	YES	prerenal	3	HD	8	2	YES	YES	PR	CR	NO	NO	no	yes	NO
66	N.J.K.	193851	NO	YES	NO	postrenal	3	ND	21	0	NO	NO	PR	CR	YES	NO	no	no	NO
67	A.S.	449931	YES	NO	NO	prerenal	1	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
68	M.R.	449952	NO	YES	NO	renal	3	SLED	14	10	YES	YES	PR	PR	YES	NO	yes	no	YES
69	V.D.	430907	NO	NO	YES	renal	3	ND	5	0	NO	NO	CR	CR	NO	NO	no	yes	NO
70	S.S.	449953	NO	YES	NO	renal	3	ND	15	8	YES	NO	PR	CR	NO	NO	no	no	NO
71	K.L.	448313	YES	NO	NO	renal	2	ND	7	0	NO	NO	PR	PR	NO	NO	no	yes	NO
72	S.C.B.	449979	NO	YES	NO	renal	3	SLED	14	9	YES	YES	PR	CR	YES	NO	no	no	NO
73	K.L.	450006	NO	YES	NO	postrenal	2	ND	6	0	NO	NO	CR	CR	NO	NO	no	no	NO
74	S.D.	449273	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
75	L.S.	166568	NO	YES	NO	postrenal	3	ND	11	0	NO	NO	PR	PR	YES	NO	no	yes	NO
76	V.S.	450020	NO	YES	NO	postrenal	3	ND	8	0	NO	NO	PR	PR	YES	NO	no	no	NO

S.no.	Name	MRD No.	Age	Gender	Rural/ Urban	Comorbidities	Comorbidity COAD	Comorbidity CLD	Comorbidity DM	Comorbidity HTN	Comorbidity CKD known	Comorbidity CKD (total)	Comorbidity CAD	Comorbidity HCV	Comorbidity HBV	Comorbidity HIV	Comorbidity MALIGNANCY	Comorbidity NEPHROLITHIASIS	Comorbidity TB	Comorbidity RA	Comorbidity CVA	Primary Etiology OF AKI	MULTIFACTORIAL AKI	ETIOLOGY GROUP
77	T.P.V.	439751	24	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	GLOMERULONEPHRITIS	NO	GLOMERULONEPHRITIS
78	I.K.	450025	65	F	R	≥3	NO	YES	YES	YES	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	? DRUG- CAM	NO	DRUG
79	A.S.	113785	63	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	HYPERVITAMINOSIS D	NO	DRUG
80	J.K.	86880	27	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREECLAMPSIA	NO	PREECLAMPSIA
81	V.G.	405882	45	M	R	2	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	DRUGS-RIFAMPICIN	NO	DRUG
82	I.K.	439615	23	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREPUREAL SEPSIS/IUD	NO	SEPSIS
83	G.K.	397112	51	F	R	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	NO	DRUG
84	Y.S.	449079	21	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	PARAQUOT POISONING	NO	POISONING
85	H.S.	431493	20	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
86	M.S.	534547	56	M	U	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
87	A.S.	534510	37	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
88	N.D.	566071	46	F	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SNAKE BITE	NO	SNAKE BITE
89	T.R.	567615	21	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	GLOMERULONEPHRITIS	NO	GLOMERULONEPHRITIS
90	K.J.K.	506487	40	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	ACUTE PANCREATITIS	NO	HYPOVOLEMIA
91	H.K.	568798	34	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
92	P.S.	262623	51	M	U	2	NO	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-SEPSIS/D.FOOT	NO	SEPSIS
93	L.A.	553879	57	M	R	≥3	NO	NO	YES	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	SUR-PAPILLARY NECROSIS	NO	U.O.
94	E.B.	490445	35	M	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	? SEPSIS/FOCUS NK	NO	SEPSIS
95	S.B.	562559	41	M	R	2	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
96	N.S.	558879	19	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OPC POISONING	NO	POISONING
97	B.S.	559084	39	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
98	R.K.P.	558842	58	M	U	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG-ACYCLOVIR	NO	DRUG
99	J.S.	522475	27	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
100	B.S.	552073	65	M	R	≥3	NO	NO	NO	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	YES	CARDIORENAL
101	P.S.	553057	43	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
102	T.S.	249300	64	M	R	≥3	YES	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
103	S.S.	549094	42	M	U	1	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/GASTROENTERITIS	NO	SEPSIS
104	M.B.	47391	43	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
105	B.S.	384116	46	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- ARB	NO	DRUG
106	S.D.	517992	47	F	U	≥3	NO	NO	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	SEPSIS/PYELONEPHRITIS	YES	SEPSIS
107	A.S.	515593	49	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/MENINGITIS	NO	SEPSIS
108	S.R.	541455	23	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREPUREAL SEPSIS	NO	SEPSIS
109	N.K.	535414	30	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREPUREAL SEPSIS	NO	SEPSIS
110	G.S.	517952	55	M	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
111	G.S.	543717	62	M	U	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	SUR-Ca BLADDER	NO	U.O.
112	S.S.	496767	63	M	U	≥3	NO	NO	YES	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	NO	DRUG
113	S.S.	498419	65	M	R	≥3	NO	YES	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-BPH	NO	U.O.
114	S.D.	573340	39	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SNAKE BITE	NO	SNAKE BITE
115	J.K.	570314	29	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	HYPERVITAMINOSIS D	NO	DRUG
116	J.S.	513907	43	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-LEPTO	NO	TROPICAL FEVER
117	S.S.	507519	47	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
118	T.S.	512180	48	M	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
119	S.R.	508234	49	F	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- ARB	YES	DRUG
120	A.S.	481320	42	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
121	D.S.	267354	40	M	R	2	NO	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-SEPSIS/D.FOOT	NO	SEPSIS
122	S.S.	488421	59	M	R	2	NO	YES	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/SBP	YES	SEPSIS
123	B.D.	487742	52	M	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
124	M.P.	487436	20	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	aHUS	NO	HUS
125	C.S.	479558	41	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-MALARIA	NO	TROPICAL FEVER
126	M.S.	440867	48	M	R	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	YES	SEPSIS
127	M.R.	474984	42	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-ENTERIC	NO	TROPICAL FEVER
128	B.S.	469124	59	M	U	≥3	YES	NO	YES	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	YES	CARDIORENAL
129	K.G.	474939	57	F	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
130	K.D.	471336	53	F	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG-ACYCLOVIR	NO	DRUG
131	S.S.	465025	64	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	MULTIPLE MYELOMA	NO	MULTIPLE MYELOMA
132	R.D.	437699	51	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	GLOMERULONEPHRITIS	NO	GLOMERULONEPHRITIS
133	A.S.	457880	54	M	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	TROPICAL-ENTERIC	NO	TROPICAL FEVER
134	S.K.	45609	52	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/GASTROENTERITIS	YES	SEPSIS
135	K.I.	453830	26	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
136	G.S.	59298	48	M	R	≥3	NO	NO	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	YES	NO	YES	SEPSIS/PNEUMONIA	YES	SEPSIS
137	J.S.W.	428557	62	M	U	2	NO	YES	NO	YES	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	TROPICAL-ENTERIC	NO	TROPICAL FEVER
138	R.M.	421485	50	M	R	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	YES	DRUG
139	K.L.	456175	52	M	U	2	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- ARB	NO	DRUG
140	L.S.	463344	54	M	R	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/COLITIS	NO	SEPSIS
141	P.R.	468079	31	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
142	N.K.	467971	53	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
143	K.S.	460657	53	M	R	1	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	HEMORRHAGIC SHOCK	NO	HYPOVOLEMIA
144	S.S.	468714	52	M	R	≥3	NO	NO	YES	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	SUR-SEPSIS/D.FOOT	NO	SEPSIS
145	R.P.R.	460781	53	M	U	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-SEPSIS/FOURNIER'S GANGRENE	NO	SEPSIS
146	M.S.	469715	51	M	R	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
147	R.P.	269376	43	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
148	G.K.	422976	46	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-ENTERIC	NO	TROPICAL FEVER
149	R.S.	443525	54	M	R	2	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	A.G.E	NO	HYPOVOLEMIA
150	V.K.	456935	27	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
151	D.P.S.	468825	43	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-ENTERIC	NO	TROPICAL FEVER
152	V.B.	419135	47	M	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	? SEPSIS/FOCUS NK	YES	SEPSIS

S.no.	Name	MRD No.	Medical AKI	Surgical AKI	Obstetric AKI	TYPE OF AKI	stage	Initial dialysis modality	Hospital stay	ICU Stay	HYPOTENSION	OLIGURIA	outcome discharge	Outcome 1 month	Readmission	Biopsy	Infective	Tropical etiology	Need of inotropes
77	T.P.V.	439751	YES	NO	NO	renal	3	ND	9	0	NO	NO	NR	PR	YES	RPGN-VASCULITIS	no	no	NO
78	I.K.	450025	YES	NO	NO	renal	3	HD	12	0	NO	YES	PR	DEATH,20	YES	NO	no	yes	NO
79	A.S.	113785	YES	NO	NO	renal	3	ND	7	0	NO	NO	PR	PR	NO	NO	no	yes	NO
80	J.K.	86880	NO	NO	YES	renal	3	ND	8	0	NO	NO	PR	PR	NO	NO	no	yes	NO
81	V.G.	405882	YES	NO	NO	renal	2	ND	10	0	NO	NO	PR	PR	NO	AIN	no	yes	NO
82	I.K.	439615	NO	NO	YES	renal	3	CRRT	2	2	YES	NO	DEATH,2	DEATH,2	NA	NO	yes	yes	YES
83	G.K.	397112	YES	NO	NO	renal	3	ND	9	0	NO	NO	NR	NR	YES	AIN	no	yes	NO
84	Y.S.	449079	YES	NO	NO	renal	3	HD	16	16	NO	YES	DEATH,16	DEATH,16	NA	NO	no	yes	NO
85	H.S.	431493	NO	YES	NO	renal	3	ND	13	7	YES	NO	PR	PR	NO	NO	no	no	NO
86	M.S.	534547	NO	YES	NO	renal	3	HD	17	11	YES	YES	PR	CR	YES	NO	no	no	NO
87	A.S.	534510	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
88	N.D.	566071	YES	NO	NO	renal	3	CRRT	16	11	YES	YES	NR	DEATH,28	YES	NO	no	yes	YES
89	T.R.	567615	YES	NO	NO	renal	2	ND	12	0	NO	NO	PR	CR	NO	IRGN	no	no	NO
90	K.J.K.	506487	YES	NO	NO	prerenal	3	SLED	14	9	YES	YES	PR	PR	YES	ATN	no	no	NO
91	H.K.	568798	YES	NO	NO	prerenal	1	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
92	P.S.	262623	NO	YES	NO	renal	3	PD	5	5	YES	NO	DEATH,5	DEATH,5	NA	NO	yes	no	YES
93	L.A.	553879	NO	YES	NO	postrenal	3	ND	9	0	NO	NO	PR	PR	YES	NO	no	no	NO
94	E.B.	490445	YES	NO	NO	renal	2	ND	9	0	NO	NO	PR	CR	NO	NO	yes	no	NO
95	S.B.	562559	YES	NO	NO	renal	3	ND	13	9	YES	NO	PR	PR	YES	NO	yes	no	YES
96	N.S.	558879	YES	NO	NO	prerenal	3	ND	16	13	YES	NO	CR	CR	NO	NO	no	yes	YES
97	B.S.	559084	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
98	R.K.P.	558842	YES	NO	NO	renal	2	ND	6	0	NO	NO	PR	PR	NO	NO	no	no	NO
99	J.S.	522475	NO	YES	NO	postrenal	3	ND	6	0	NO	NO	NR	CR	YES	NO	no	yes	NO
100	B.S.	552073	YES	NO	NO	prerenal	1	ND	6	0	YES	NO	PR	CR	NO	NO	no	no	NO
101	P.S.	553057	YES	NO	NO	prerenal	2	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
102	T.S.	249300	YES	NO	NO	renal	3	SLED	10	7	YES	YES	NR	NR	YES	NO	yes	no	YES
103	S.S.	549094	YES	NO	NO	prerenal	3	ND	8	0	YES	NO	PR	CR	NO	NO	yes	no	YES
104	M.B.	47391	YES	NO	NO	prerenal	1	ND	5	0	YES	NO	CR	CR	NO	NO	no	yes	NO
105	B.S.	384116	YES	NO	NO	renal	2	ND	4	0	NO	NO	CR	CR	NO	NO	no	no	NO
106	S.D.	517992	YES	NO	NO	renal	2	ND	5	0	NO	YES	DEATH,5	DEATH,5	NA	NO	yes	no	NO
107	A.S.	515593	YES	NO	NO	renal	3	PD	21	16	YES	YES	PR	PR	YES	NO	yes	no	YES
108	S.R.	541455	NO	NO	YES	renal	3	ND	3	0	NO	NO	DEATH,3	DEATH,3	NA	NO	yes	yes	NO
109	N.K.	535414	NO	NO	YES	renal	3	ND	9	3	YES	NO	PR	CR	NO	NO	yes	yes	YES
110	G.S.	517952	YES	NO	NO	prerenal	2	ND	10	0	YES	NO	PR	CR	NO	NO	no	yes	NO
111	G.S.	543717	NO	YES	NO	postrenal	3	ND	12	0	NO	NO	PR	PR	YES	NO	no	no	NO
112	S.S.	496767	YES	NO	NO	renal	3	HD	14	0	NO	YES	NR	DEATH,23	YES	NO	no	yes	NO
113	S.S.	498419	NO	YES	NO	postrenal	3	ND	5	0	NO	NO	PR	CR	YES	NO	no	no	NO
114	S.D.	573340	YES	NO	NO	renal	3	HD	25	0	NO	YES	NR	NR	YES	AIN	no	yes	NO
115	J.K.	570314	YES	NO	NO	renal	3	ND	5	0	NO	NO	PR	PR	NO	NO	no	yes	NO
116	J.S.	513907	YES	NO	NO	renal	2	ND	8	2	NO	NO	PR	CR	NO	NO	yes	yes	NO
117	S.S.	507519	YES	NO	NO	prerenal	2	ND	5	0	YES	NO	PR	CR	NO	NO	no	yes	NO
118	T.S.	512180	YES	NO	NO	prerenal	1	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
119	S.R.	508234	YES	NO	NO	renal	1	ND	3	0	NO	NO	CR	CR	NO	NO	no	no	NO
120	A.S.	481320	YES	NO	NO	prerenal	2	ND	5	0	YES	NO	PR	CR	NO	NO	no	yes	NO
121	D.S.	267354	NO	YES	NO	renal	3	SLED	4	4	YES	NO	DEATH,4	DEATH,4	NA	NO	yes	no	YES
122	S.S.	488421	YES	NO	NO	prerenal	3	PD	2	2	YES	YES	DEATH,2	DEATH,2	NA	NO	yes	no	YES
123	B.D.	487742	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
124	M.P.	487436	YES	NO	NO	renal	3	HD	16	0	NO	YES	NR	NR	YES	TMA	no	no	NO
125	C.S.	479558	YES	NO	NO	renal	2	ND	6	3	YES	NO	PR	CR	NO	NO	yes	yes	NO
126	M.S.	440867	YES	NO	NO	renal	3	PD	4	4	YES	NO	DEATH,4	DEATH,4	NA	NO	yes	no	YES
127	M.R.	474984	YES	NO	NO	prerenal	3	SLED	7	3	YES	NO	PR	CR	YES	NO	yes	yes	NO
128	B.S.	469124	YES	NO	NO	prerenal	2	ND	12	5	YES	NO	PR	PR	NO	NO	no	no	YES
129	K.G.	474939	YES	NO	NO	prerenal	1	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
130	K.D.	471336	YES	NO	NO	renal	2	ND	5	0	NO	NO	PR	PR	NO	NO	no	no	NO
131	S.S.	465025	YES	NO	NO	renal	3	HD	13	0	NO	YES	NR	NR	YES	CAST NEPH	no	no	NO
132	R.D.	437699	YES	NO	NO	renal	3	ND	9	0	NO	YES	NR	PR	YES	ANTI-GBM	no	no	NO
133	A.S.	457880	YES	NO	NO	prerenal	1	ND	6	0	NO	NO	PR	CR	NO	NO	yes	yes	NO
134	S.K.	45609	YES	NO	NO	prerenal	2	ND	11	3	NO	NO	PR	CR	NO	NO	yes	no	NO
135	K.I.	453830	NO	YES	NO	renal	3	ND	21	12	YES	NO	CR	CR	NO	NO	no	no	NO
136	G.S.	59298	YES	NO	NO	renal	3	HD	10	3	NO	YES	DEATH,10	DEATH,10	NA	NO	yes	no	NO
137	J.S.W.	428557	YES	NO	NO	prerenal	2	ND	9	3	NO	NO	DEATH,9	DEATH,9	NA	NO	yes	yes	NO
138	R.M.	421485	YES	NO	NO	renal	2	ND	9	0	NO	NO	NR	NR	NO	AIN	no	yes	NO
139	K.L.	456175	YES	NO	NO	renal	3	ND	5	0	NO	NO	PR	PR	NO	NO	no	no	NO
140	L.S.	463344	YES	NO	NO	renal	3	PD	9	6	YES	YES	NR	NR	YES	NO	yes	no	YES
141	P.R.	468079	YES	NO	NO	renal	3	ND	7	0	YES	NO	PR	PR	NO	NO	yes	no	YES
142	N.K.	467971	YES	NO	NO	renal	3	SLED	5	5	YES	NO	DEATH,5	DEATH,5	NA	NO	yes	no	YES
143	K.S.	460657	YES	NO	NO	prerenal	3	ND	14	11	YES	NO	PR	PR	NO	NO	no	no	NO
144	S.S.	468714	NO	YES	NO	renal	3	HD	16	3	YES	YES	NR	NR	YES	NO	yes	no	YES
145	R.P.R.	460781	NO	YES	NO	renal	3	SLED	5	5	YES	NO	DEATH,5	DEATH,5	NA	NO	yes	no	YES
146	M.S.	469715	YES	NO	NO	renal	3	PD	3	3	YES	YES	DEATH,3	DEATH,3	NA	NO	yes	no	YES
147	R.P.	269376	YES	NO	NO	prerenal	2	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
148	G.K.	422976	YES	NO	NO	prerenal	2	ND	7	0	YES	NO	NR	CR	NO	NO	yes	yes	NO
149	R.S.	443525	YES	NO	NO	prerenal	1	ND	3	0	YES	YES	PR	CR	NO	NO	no	yes	NO
150	V.K.	456935	NO	YES	NO	renal	3	ND	30	25	YES	NO	PR	CR	NO	NO	no	no	NO
151	D.P.S.	468825	YES	NO	NO	prerenal	2	ND	8	0	NO	NO	CR	CR	NO	NO	yes	yes	NO
152	V.B.	419135	YES	NO	NO	renal	3	HD	15	0	YES	YES	NR	NR	NO	NO	yes	no	YES

S.no.	Name	MRD No.	Age	Gender	Rural/ Urban	Comorbidities	Comorbidity COAD	Comorbidity CLD	Comorbidity DM	Comorbidity HTN	Comorbidity CKD known	Comorbidity CKD (total)	Comorbidity CAD	Comorbidity HCV	Comorbidity HBV	Comorbidity HIV	Comorbidity MALIGNANCY	Comorbidity NEPHROLITHIASIS	Comorbidity TB	Comorbidity RA	Comorbidity CVA	Primary Etiology OF AKI	MULTIFACTORIAL AKI	ETIOLOGY GROUP
153	S.C.S.	451051	49	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/GASTROENTERITIS	NO	SEPSIS
154	P.K.	454441	53	F	R	2	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
155	G.K.	464269	51	F	R	2	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
156	J.K.	159326	48	F	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	? SEPSIS/FOCUS NK	NO	SEPSIS
157	S.G.	464146	40	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	ACUTE PANCREATITIS	NO	HYPOVOLEMIA
158	I.K.	469252	56	F	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
159	T.S.	469980	57	F	U	≥ 3	YES	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	NO	DRUG
160	P.K.	465550	46	F	R	2	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
161	B.K.	466363	39	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
162	N.D.	441156	40	F	R	2	NO	NO	NO	YES	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
163	G.K.	102480	43	F	R	2	YES	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
164	V.W.	468039	51	F	R	1	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
165	A.K.	469905	21	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SNAKE BITE	NO	SNAKE BITE
166	G.S.	470013	48	M	R	2	NO	NO	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	DRUG- NSAIDS	YES	DRUG
167	S.K.	269590	51	F	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	A.G.E	NO	HYPOVOLEMIA
168	S.K.	467213	49	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-ENTERIC	NO	TROPICAL FEVER
169	K.L.	456175	39	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	ETHYLENE GLYCOL POISONING	NO	POISONING
170	P.K.	414842	38	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
171	A.K.	469136	61	F	R	≥ 3	NO	NO	NO	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
172	S.K.	469457	62	M	R	≥ 3	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	YES	SEPSIS
173	K.S.	470051	19	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	GLOMERULONEPHRITIS	NO	GLOMERULONEPHRITIS
174	S.K.B.	470202	64	M	U	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-ENTERIC	NO	TROPICAL FEVER
175	R.S.	470655	42	M	R	≥ 3	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	DRUG- NSAIDS	YES	DRUG
176	B.S.	475460	43	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
177	A.M.	394527	46	M	R	1	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	HRS	NO	HRS
178	S.S.	476883	47	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
179	A.D.	451107	49	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-SEPSIS/BURNS	NO	SEPSIS
180	G.S.	479292	40	M	R	1	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
181	N.S.	480094	38	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-LEPTO	NO	TROPICAL FEVER
182	S.K.	480103	55	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	NO	CARDIORENAL
183	G.K.	359285	62	F	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-MALARIA	NO	TROPICAL FEVER
184	G.S.	481221	34	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SNAKE BITE	NO	SNAKE BITE
185	A.C.	159793	45	M	U	1	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	LEAD POISONING	NO	POISONING
186	A.K.G.	482162	39	M	U	≥ 3	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	YES	NO	NO	DRUG- TENOFOVIR	NO	DRUG
187	S.K.	482273	41	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
188	K.S.	482763	43	M	R	2	NO	YES	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	ACUTE PANCREATITIS	NO	HYPOVOLEMIA
189	B.K.	482891	19	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	PARAQUOT POISONING	NO	POISONING
190	A.G.	450033	48	M	U	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	MERCURY POISONING	NO	POISONING
191	S.G.	478599	63	F	R	≥ 3	NO	NO	NO	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/GASTROENTERITIS	YES	SEPSIS
192	V.J.	150463	62	M	R	≥ 3	NO	NO	NO	YES	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	YES	CARDIORENAL
193	G.S.	483455	60	M	R	2	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-PAPILLARY NECROSIS	NO	U.O.
194	H.G.	483556	39	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-ENTERIC	NO	TROPICAL FEVER
195	S.K.	483653	39	F	U	1	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	YES	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
196	H.L.G.	483665	40	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
197	G.S.	483692	41	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
198	D.S.	483719	43	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
199	K.R.	483836	49	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-LEPTO	NO	TROPICAL FEVER
200	J.S.	484331	48	M	R	2	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	SUR-NEUROGENIC BLADDER	NO	U.O.
201	V.A.	368768	42	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
202	C.K.	78742	47	F	R	1	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
203	J.K.	389549	51	F	U	1	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
204	R.D.	430756	48	F	U	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
205	K.W.	306178	56	F	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
206	R.D.	431329	38	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-MALARIA	NO	TROPICAL FEVER
207	K.R.	502410	59	F	R	1	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
208	K.K.	506487	50	F	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	NO	DRUG
209	N.S.	502354	52	F	R	2	NO	YES	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/SBP	YES	SEPSIS
210	K.S.	491072	55	M	U	1	NO	NO	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	YES	SEPSIS
211	K.V.	433875	39	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	PARAQUOT POISONING	NO	POISONING
212	M.L.	507473	40	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
213	S.S.	509752	47	M	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/MENINGITIS	NO	SEPSIS
214	N.V.	508304	52	M	U	0	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
215	S.S.	501634	53	M	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	A.G.E	NO	HYPOVOLEMIA
216	R.S.C.	504153	34	M	R	1	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/GASTROENTERITIS	YES	SEPSIS
217	D.K.	508231	39	F	R	1	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
218	P.J.	508347	41	M	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
219	B.L.	509310	43	M	U	2	NO	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	YES	DRUG
220	R.R.	502365	51	F	R	2	NO	YES	YES	NO	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	? DRUG- CAM	NO	DRUG
221	U.G.	290888	56	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
222	K.R.	507309	57	M	R	1	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
223	B.S.	510298	65	M	R	2	NO	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	NO	CARDIORENAL
224	P.S.	502412	64	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-BPH	NO	U.O.
225	S.S.	455877	27	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E, CHOLERA	NO	HYPOVOLEMIA
226	R.K.	510570	60	M	U	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-BPH	NO	U.O.
227	G.K.	186184	36	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
228	D.K.	484462	63	F	R	≥ 3	NO	NO	YES	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	YES	LVF/CARDIORENAL	NO	CARDIORENAL

S.no.	Name	MRD No.	Medical AKI	Surgical AKI	Obstetric AKI	TYPE OF AKI	stage	Initial dialysis modality	Hospital stay	ICU Stay	HYPOTENSION	OLIGURIA	outcome discharge	Outcome 1 month	Readmission	Biopsy	Infective	Tropical etiology	Need of inotropes
153	S.C.S.	451051	YES	NO	NO	prerenal	3	HD	10	0	YES	YES	PR	CR	NO	NO	yes	no	YES
154	P.K.	454441	YES	NO	NO	renal	2	ND	7	0	YES	NO	PR	PR	NO	NO	yes	no	YES
155	G.K.	464269	NO	YES	NO	postrenal	2	ND	11	0	NO	NO	CR	CR	NO	NO	no	yes	NO
156	J.K.	159326	YES	NO	NO	renal	3	CRRT	5	5	YES	YES	DEATH,5	DEATH,5	NA	NO	yes	no	YES
157	S.G.	464146	YES	NO	NO	prerenal	2	ND	18	7	YES	YES	NR	CR	NO	NO	no	no	NO
158	I.K.	469252	YES	NO	NO	prerenal	2	ND	6	0	YES	YES	PR	CR	NO	NO	no	yes	NO
159	T.S.	469980	YES	NO	NO	renal	3	ND	9	0	NO	NO	NR	NR	YES	AIN	no	yes	NO
160	P.K.	465550	YES	NO	NO	renal	3	PD	12	8	YES	YES	PR	PR	YES	NO	yes	no	YES
161	B.K.	466363	NO	YES	NO	renal	3	ND	16	9	YES	NO	PR	CR	NO	NO	no	no	NO
162	N.D.	441156	NO	YES	NO	postrenal	3	ND	6	0	NO	NO	PR	PR	NO	NO	no	yes	NO
163	G.K.	102480	YES	NO	NO	renal	3	SLED	9	4	YES	YES	PR	PR	YES	NO	yes	no	YES
164	V.W.	468039	YES	NO	NO	prerenal	1	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
165	A.K.	469905	YES	NO	NO	renal	3	HD	15	0	NO	YES	CR	CR	NO	NO	no	yes	NO
166	G.S.	470013	YES	NO	NO	renal	3	SLED	18	15	YES	YES	NR	DEATH,26	YES	NO	no	yes	YES
167	S.K.	269590	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
168	S.K.	467213	YES	NO	NO	prerenal	2	ND	6	0	NO	NO	CR	CR	NO	NO	yes	yes	NO
169	K.L.	456175	YES	NO	NO	renal	3	HD	8	8	NO	YES	DEATH,8	DEATH,8	NA	NO	no	yes	NO
170	P.K.	414842	YES	NO	NO	prerenal	2	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
171	A.K.	469136	YES	NO	NO	renal	3	SLED	6	6	YES	YES	DEATH,6	DEATH,6	NA	NO	yes	no	YES
172	S.K.	469457	YES	NO	NO	renal	3	HD	16	0	YES	YES	NR	PR	YES	NO	yes	no	YES
173	K.S.	470051	YES	NO	NO	renal	3	ND	15	3	NO	NO	PR	CR	NO	IRGN	no	no	NO
174	S.K.B.	470202	YES	NO	NO	prerenal	3	ND	15	5	NO	NO	DEATH,15	DEATH,15	NA	NO	yes	yes	NO
175	R.S.	470655	YES	NO	NO	renal	3	HD	16	7	NO	YES	NR	NR	YES	NO	no	yes	NO
176	B.S.	475460	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
177	A.M.	394527	YES	NO	NO	prerenal	3	SLED	2	2	YES	YES	DEATH,2	DEATH,2	NA	NO	no	no	YES
178	S.S.	476883	YES	NO	NO	prerenal	1	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
179	A.D.	451107	NO	YES	NO	prerenal	3	SLED	5	5	YES	YES	DEATH,5	DEATH,5	NA	NO	yes	no	YES
180	G.S.	479292	NO	YES	NO	postrenal	3	ND	7	0	NO	NO	PR	PR	NO	NO	no	yes	NO
181	N.S.	480094	YES	NO	NO	renal	2	ND	13	5	YES	NO	PR	CR	NO	NO	yes	yes	NO
182	S.K.	480103	YES	NO	NO	prerenal	2	ND	5	0	YES	NO	PR	PR	NO	NO	no	no	YES
183	G.K.	359285	YES	NO	NO	renal	2	ND	5	0	NO	NO	PR	CR	NO	NO	yes	yes	NO
184	G.S.	481221	YES	NO	NO	renal	3	SLED	15	15	YES	YES	NR	DEATH,19	YES	NO	no	yes	YES
185	A.C.	159793	YES	NO	NO	renal	2	ND	8	0	YES	NO	NR	NR	NO	NO	no	yes	NO
186	A.K.G.	482162	YES	NO	NO	renal	3	HD	11	3	NO	YES	DEATH,11	DEATH,11	NA	NO	no	no	NO
187	S.K.	482273	YES	NO	NO	renal	3	HD	8	0	YES	YES	PR	PR	NO	NO	yes	no	YES
188	K.S.	482763	YES	NO	NO	prerenal	3	HD	14	3	NO	YES	PR	DEATH,21	YES	NO	no	no	NO
189	B.K.	482891	YES	NO	NO	renal	3	HD	17	17	NO	YES	DEATH,17	DEATH,17	NA	NO	no	yes	NO
190	A.G.	450033	YES	NO	NO	renal	3	HD	9	0	NO	YES	NR	PR	YES	NO	no	yes	NO
191	S.G.	478599	YES	NO	NO	prerenal	3	SLED	4	4	YES	NO	DEATH,4	DEATH,4	NA	NO	yes	no	YES
192	V.J.	150463	YES	NO	NO	prerenal	2	ND	7	0	YES	NO	PR	PR	NO	NO	no	no	YES
193	G.S.	483455	NO	YES	NO	postrenal	3	ND	11	3	NO	NO	PR	CR	YES	NO	no	no	NO
194	H.G.	483556	YES	NO	NO	prerenal	2	ND	6	0	NO	NO	PR	CR	NO	NO	yes	yes	NO
195	S.K.	483653	NO	YES	NO	postrenal	3	ND	10	0	NO	NO	PR	PR	NO	NO	no	yes	NO
196	H.L.G.	483665	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
197	G.S.	483692	YES	NO	NO	prerenal	1	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
198	D.S.	483719	YES	NO	NO	prerenal	2	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
199	K.R.	483836	YES	NO	NO	renal	2	ND	5	0	NO	NO	PR	CR	NO	NO	yes	yes	NO
200	J.S.	484331	NO	YES	NO	postrenal	2	ND	13	0	NO	NO	NR	PR	NO	NO	no	no	NO
201	V.A.	368768	YES	NO	NO	prerenal	1	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
202	C.K.	78742	YES	NO	NO	prerenal	2	ND	6	0	YES	YES	PR	CR	NO	NO	no	yes	NO
203	J.K.	389549	YES	NO	NO	prerenal	2	ND	5	0	YES	YES	PR	CR	NO	NO	no	yes	NO
204	R.D.	430756	YES	NO	NO	renal	3	PD	5	5	YES	NO	DEATH,5	DEATH,5	NA	NO	yes	no	YES
205	K.W.	306178	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
206	R.D.	431329	YES	NO	NO	renal	2	ND	6	0	NO	NO	PR	CR	NO	NO	yes	yes	NO
207	K.R.	502410	YES	NO	NO	prerenal	3	ND	11	5	NO	NO	NR	PR	YES	NO	yes	yes	NO
208	K.K.	506487	YES	NO	NO	renal	3	ND	8	0	NO	NO	NR	NR	YES	AIN	no	yes	NO
209	N.S.	502354	YES	NO	NO	prerenal	3	SLED	6	6	YES	YES	DEATH,6	DEATH,6	NA	NO	yes	no	YES
210	K.S.	491072	YES	NO	NO	renal	3	ND	12	4	YES	NO	PR	PR	NO	NO	yes	no	NO
211	K.V.	433875	YES	NO	NO	renal	3	HD	12	12	NO	YES	DEATH,12	DEATH,12	NA	NO	no	yes	NO
212	M.L.	507473	YES	NO	NO	prerenal	2	ND	7	0	NO	NO	PR	CR	NO	NO	yes	yes	NO
213	S.S.	509752	YES	NO	NO	renal	3	PD	6	6	YES	YES	DEATH,6	DEATH,6	NA	NO	yes	no	YES
214	N.V.	508304	YES	NO	NO	renal	2	ND	12	4	NO	NO	PR	PR	NO	NO	yes	no	NO
215	S.S.	501634	YES	NO	NO	prerenal	1	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
216	R.S.C.	504153	YES	NO	NO	prerenal	3	PD	15	10	YES	YES	PR	PR	YES	NO	yes	no	YES
217	D.K.	508231	YES	NO	NO	prerenal	2	ND	7	0	YES	YES	PR	CR	NO	NO	no	yes	NO
218	P.J.	508347	YES	NO	NO	prerenal	2	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
219	B.L.	509310	YES	NO	NO	renal	2	ND	16	0	NO	NO	NR	PR	NO	NO	no	no	NO
220	R.R.	502365	YES	NO	NO	renal	3	HD	14	0	NO	YES	NR	DEATH,25	YES	NO	no	yes	NO
221	U.G.	290888	YES	NO	NO	prerenal	3	ND	14	4	NO	NO	PR	CR	YES	NO	yes	yes	NO
222	K.R.	507309	YES	NO	NO	renal	3	SLED	10	5	YES	YES	PR	PR	YES	NO	yes	no	NO
223	B.S.	510298	YES	NO	NO	prerenal	2	ND	7	0	YES	NO	PR	PR	NO	NO	no	no	NO
224	P.S.	502412	NO	YES	NO	postrenal	3	ND	8	0	NO	NO	NR	CR	YES	NO	no	no	NO
225	S.S.	455877	YES	NO	NO	prerenal	2	ND	8	0	YES	NO	PR	CR	NO	NO	no	yes	NO
226	R.K.	510570	NO	YES	NO	postrenal	2	ND	5	0	NO	NO	CR	CR	NO	NO	no	no	NO
227	G.K.	186184	YES	NO	NO	prerenal	1	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
228	D.K.	484462	YES	NO	NO	prerenal	3	SLED	8	7	YES	YES	NR	DEATH,15	YES	NO	no	no	YES

S.no.	Name	MRD No.	Age	Gender	Rural/ Urban	Comorbidities	Comorbidity COAD	Comorbidity CLD	Comorbidity DM	Comorbidity HTN	Comorbidity CKD known	Comorbidity CKD (total)	Comorbidity CAD	Comorbidity HCV	Comorbidity HBV	Comorbidity HIV	Comorbidity MALIGNANCY	Comorbidity NEPHROLITHIASIS	Comorbidity TB	Comorbidity RA	Comorbidity CVA	Primary Etiology OF AKI	MULTIFACTORIAL AKI	ETIOLOGY GROUP
229	S.S.	496767	52	M	U	1	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/GASTROENTERITIS	YES	SEPSIS
230	B.S.	481486	39	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E, CHOLERA	NO	HYPOVOLEMIA
231	B.C.	527116	40	M	R	> 3	NO	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	YES	SUR-SEPSIS/D.FOOT	YES	SEPSIS
232	H.C.J.	527787	47	M	R	2	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
233	K.D.	528461	52	F	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
234	S.K.	527174	53	M	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
235	A.D.K.	525544	46	F	R	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
236	M.K.	527478	39	F	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
237	K.S.	520072	41	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
238	R.S.S.	528154	43	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
239	P.D.	527816	44	F	U	1	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
240	S.S.	527916	56	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
241	G.S.	520095	57	M	R	1	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
242	R.S.	528609	65	M	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	SUR-Ca PROSTATE	NO	U.O.
243	S.S.	528375	34	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	ACUTE PANCREATITIS	NO	HYPOVOLEMIA
244	K.K.	527190	61	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
245	A.K.	526750	27	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/URETHRAL INJURY	NO	U.O.
246	M.P.	504912	42	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
247	J.S.	529026	63	M	U	1	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	YES	CARDIORENAL
248	R.D.	527080	52	M	R	1	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
249	M.S.	529688	51	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/GASTROENTERITIS	NO	SEPSIS
250	S.A.	527822	56	M	R	2	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	TROPICAL-SCRUB	NO	TROPICAL FEVER
251	S.P.D.	518703	50	M	U	2	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
252	B.K.	465261	53	F	U	1	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
253	S.M.	525458	54	F	R	2	NO	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	NO	CARDIORENAL
254	C.W.	524276	37	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
255	A.B.	525456	38	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-LEPTO	NO	TROPICAL FEVER
256	A.G.	527368	30	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
257	H.S.	523054	40	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SNAKE BITE	NO	SNAKE BITE
258	T.S.	529019	45	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
259	P.S.	527126	46	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
260	L.D.	520047	48	F	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
261	S.D.	547210	38	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
262	M.K.	550807	40	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
263	A.K.	546389	43	F	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
264	K.D.	531558	46	F	R	1	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
265	D.S.	546440	54	M	R	2	NO	YES	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/SBP	YES	SEPSIS
266	G.K.	547208	59	F	U	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	GLOMERULONEPHRITIS	NO	GLOMERULONEPHRITIS
267	S.D.	189603	32	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREECLAMPSIA	NO	PREECLAMPSIA
268	S.R.	550855	66	F	U	≥ 3	YES	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- ACEI	NO	DRUG
269	S.D.	549798	52	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/GASTROENTERITIS	NO	SEPSIS
270	D.K.	548776	41	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
271	S.G.	548921	40	M	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
272	R.K.	548785	39	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	PARAQUOT POISONING	NO	POISONING
273	P.K.	547908	51	F	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
274	R.V.	540716	21	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	GLOMERULONEPHRITIS	NO	GLOMERULONEPHRITIS
275	V.S.	548907	26	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
276	S.K.	546425	37	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
277	A.J.K.	543586	26	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREPUREAL SEPSIS/IUD	NO	SEPSIS
278	B.K.	548786	31	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREECLAMPSIA	NO	PREECLAMPSIA
279	B.D.	551447	27	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREPUREAL SEPSIS	NO	SEPSIS
280	S.K.	507059	26	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
281	N.J.	551552	30	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
282	M.S.	566181	18	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SNAKE BITE	NO	SNAKE BITE
283	K.S.	572469	20	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	METHANOL POISONING	NO	POISONING
284	M.G.	568954	41	F	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
285	N.B.	571545	33	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREECLAMPSIA	NO	PREECLAMPSIA
286	B.K.	566911	32	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
287	P.R.	570830	44	F	U	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
288	U.D.	570921	31	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREPUREAL SEPSIS	NO	SEPSIS
289	J.S.	568292	27	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/URETHRAL INJURY	NO	U.O.
290	A.J.	563420	29	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREECLAMPSIA	NO	PREECLAMPSIA
291	M.K.	570820	19	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	GLOMERULONEPHRITIS	NO	GLOMERULONEPHRITIS
292	M.S.	551509	23	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OPC POISONING	NO	POISONING
293	B.D.	550793	34	F	U	≥ 3	NO	YES	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	NO	DRUG
294	S.S.	566112	26	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
295	N.S.	550346	21	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
296	K.D.	196098	25	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-SEPTIC ABORTION	NO	SEPSIS
297	L.K.	571196	23	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	PARAQUOT POISONING	NO	POISONING
298	A.S.	567577	61	M	U	2	NO	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	SUR-BPH	NO	U.O.
299	L.K.	571694	53	F	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-MALARIA	NO	TROPICAL FEVER
300	B.S.	569696	63	M	R	1	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
301	G.S.	588907	52	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
302	K.S.	591256	53	F	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
303	J.S.	122982	51	M	R	≥ 3	NO	YES	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/SBP	YES	SEPSIS
304	B.K.	389696	43	F	R	2	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER

S.no.	Name	MRD No.	Medical AKI	Surgical AKI	Obstetric AKI	TYPE OF AKI	stage	Initial dialysis modality	Hospital stay	ICU Stay	HYPOTENSION	OLIGURIA	outcome discharge	Outcome 1 month	Readmission	Biopsy	Infective	Tropical etiology	Need of inotropes
229	S.S.	496767	YES	NO	NO	prerenal	3	ND	8	3	YES	NO	PR	CR	NO	NO	yes	no	YES
230	B.S.	481486	YES	NO	NO	prerenal	2	ND	7	0	YES	YES	PR	CR	NO	NO	no	yes	NO
231	B.C.	527116	NO	YES	NO	renal	3	SLED	14	10	YES	YES	DEATH,14	DEATH,14	NA	NO	yes	no	YES
232	H.C.J.	527787	YES	NO	NO	renal	2	ND	6	0	NO	NO	PR	PR	NO	NO	yes	no	NO
233	K.D.	528461	YES	NO	NO	prerenal	3	ND	9	0	YES	NO	NR	NR	YES	NO	yes	yes	YES
234	S.K.	527174	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
235	A.D.K.	525544	YES	NO	NO	prerenal	3	SLED	2	2	YES	YES	DEATH,2	DEATH,2	NA	NO	no	yes	YES
236	M.K.	527478	NO	YES	NO	postrenal	3	HD	7	0	NO	NO	PR	CR	NO	NO	no	yes	NO
237	K.S.	520072	YES	NO	NO	prerenal	3	SLED	4	4	YES	NO	DEATH,4	DEATH,4	NA	NO	yes	yes	YES
238	R.S.S.	528154	YES	NO	NO	prerenal	1	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
239	P.D.	527816	NO	YES	NO	postrenal	3	ND	8	0	NO	NO	NR	PR	YES	NO	no	yes	NO
240	S.S.	527916	YES	NO	NO	prerenal	2	ND	11	6	NO	NO	PR	CR	NO	NO	yes	yes	NO
241	G.S.	520095	YES	NO	NO	renal	3	PD	10	8	YES	YES	PR	PR	YES	NO	yes	no	YES
242	R.S.	528609	NO	YES	NO	postrenal	2	ND	9	0	NO	NO	PR	CR	NO	NO	no	no	NO
243	S.S.	528375	YES	NO	NO	prerenal	3	ND	13	6	YES	YES	PR	CR	NO	NO	no	no	NO
244	K.K.	527190	NO	YES	NO	renal	3	HD	25	25	NO	YES	DEATH,25	DEATH,25	NA	NO	no	no	NO
245	A.K.	526750	NO	YES	NO	postrenal	3	HD	9	0	NO	NO	CR	CR	NO	NO	no	no	NO
246	M.P.	504912	YES	NO	NO	prerenal	2	ND	2	0	YES	NO	CR	CR	NO	NO	no	yes	NO
247	J.S.	529026	YES	NO	NO	prerenal	2	ND	10	4	YES	NO	NR	PR	NO	NO	no	no	YES
248	R.D.	527080	YES	NO	NO	renal	3	ND	9	0	YES	NO	PR	PR	YES	NO	yes	no	YES
249	M.S.	529688	YES	NO	NO	prerenal	3	HD	18	0	YES	YES	NR	PR	YES	NO	yes	no	YES
250	S.A.	527822	YES	NO	NO	prerenal	2	ND	14	6	NO	NO	NR	NR	NO	NO	yes	yes	NO
251	S.P.D.	518703	YES	NO	NO	renal	2	ND	8	0	NO	NO	PR	PR	NO	NO	yes	no	NO
252	B.K.	465261	YES	NO	NO	prerenal	2	ND	5	0	YES	NO	CR	CR	NO	NO	yes	yes	NO
253	S.M.	525458	YES	NO	NO	prerenal	2	ND	11	4	YES	NO	NR	PR	NO	NO	no	no	NO
254	C.W.	524276	YES	NO	NO	prerenal	1	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
255	A.B.	525456	YES	NO	NO	renal	2	ND	9	5	NO	NO	PR	CR	NO	NO	yes	yes	NO
256	A.G.	527368	YES	NO	NO	prerenal	2	ND	8	3	YES	NO	PR	CR	NO	NO	yes	yes	NO
257	H.S.	523054	YES	NO	NO	renal	3	HD	21	0	NO	YES	NR	NR	YES	ACN	no	yes	NO
258	T.S.	529019	YES	NO	NO	prerenal	2	ND	4	0	YES	NO	CR	CR	NO	NO	no	yes	NO
259	P.S.	527126	YES	NO	NO	prerenal	3	SLED	6	2	YES	NO	DEATH,6	DEATH,6	NA	NO	yes	yes	YES
260	L.D.	520047	NO	YES	NO	renal	3	ND	16	8	YES	NO	PR	PR	NO	NO	no	no	YES
261	S.D.	547210	YES	NO	NO	prerenal	2	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
262	M.K.	550807	YES	NO	NO	prerenal	2	ND	8	2	YES	NO	PR	CR	NO	NO	yes	yes	NO
263	A.K.	546389	YES	NO	NO	prerenal	2	ND	5	0	NO	NO	CR	CR	NO	NO	yes	yes	NO
264	K.D.	531558	YES	NO	NO	renal	3	ND	16	12	YES	NO	PR	CR	NO	NO	yes	no	YES
265	D.S.	546440	YES	NO	NO	prerenal	3	PD	4	4	YES	YES	DEATH,4	DEATH,4	NA	NO	yes	no	YES
266	G.K.	547208	YES	NO	NO	renal	3	HD	10	0	NO	YES	NR	NR	YES	IRGN	no	no	NO
267	S.D.	189603	NO	NO	YES	renal	2	ND	7	3	NO	NO	PR	CR	NO	NO	no	yes	NO
268	S.R.	550855	YES	NO	NO	renal	2	ND	3	0	NO	NO	PR	PR	NO	NO	no	no	NO
269	S.D.	549798	YES	NO	NO	prerenal	2	ND	9	4	YES	NO	PR	CR	NO	NO	yes	no	YES
270	D.K.	548776	YES	NO	NO	prerenal	3	PD	9	6	YES	YES	PR	PR	YES	NO	yes	yes	NO
271	S.G.	548921	NO	YES	NO	postrenal	3	ND	9	0	NO	NO	PR	PR	NO	NO	no	yes	NO
272	R.K.	548785	YES	NO	NO	renal	3	HD	16	16	NO	NO	DEATH,16	DEATH,16	NA	NO	no	yes	NO
273	P.K.	547908	YES	NO	NO	prerenal	1	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
274	R.V.	540716	YES	NO	NO	renal	3	ND	12	0	NO	YES	PR	PR	NO	MPGN	no	no	NO
275	V.S.	548907	NO	YES	NO	renal	3	ND	12	4	YES	NO	PR	CR	NO	NO	no	no	NO
276	S.K.	546425	YES	NO	NO	prerenal	3	ND	13	11	YES	NO	PR	CR	NO	NO	yes	yes	NO
277	A.J.K.	543586	NO	NO	YES	renal	3	HD	21	3	YES	YES	NR	NR	YES	NO	yes	yes	YES
278	B.K.	548786	NO	NO	YES	renal	2	ND	8	4	NO	NO	PR	CR	NO	NO	no	yes	NO
279	B.D.	551447	NO	NO	YES	renal	3	CRRT	19	10	YES	YES	CR	CR	YES	NO	yes	yes	YES
280	S.K.	507059	NO	YES	NO	renal	3	ND	15	15	YES	NO	PR	PR	YES	NO	no	no	NO
281	N.J.	551552	YES	NO	NO	prerenal	2	ND	14	10	NO	NO	PR	CR	NO	NO	yes	yes	NO
282	M.S.	566181	YES	NO	NO	renal	3	HD	10	0	NO	YES	PR	PR	NO	ATN	no	yes	NO
283	K.S.	572469	YES	NO	NO	renal	3	HD	8	8	NO	YES	DEATH,8	DEATH,8	NA	NO	no	yes	NO
284	M.G.	568954	YES	NO	NO	prerenal	3	PD	11	8	YES	YES	PR	CR	YES	NO	yes	yes	NO
285	N.B.	571545	NO	NO	YES	renal	2	ND	6	0	NO	NO	PR	CR	NO	NO	no	yes	NO
286	B.K.	566911	NO	YES	NO	postrenal	3	ND	8	4	NO	NO	PR	PR	NO	NO	no	yes	NO
287	P.R.	570830	YES	NO	NO	prerenal	2	ND	9	5	NO	NO	PR	PR	NO	NO	yes	yes	NO
288	U.D.	570921	NO	NO	YES	renal	3	PD	5	5	YES	NO	DEATH,5	DEATH,5	NA	NO	yes	yes	YES
289	J.S.	568292	NO	YES	NO	postrenal	2	ND	14	0	NO	NO	CR	CR	NO	NO	no	no	NO
290	A.J.	563420	NO	NO	YES	renal	2	ND	7	0	NO	NO	CR	CR	NO	NO	no	yes	NO
291	M.K.	570820	YES	NO	NO	renal	3	HD	9	0	NO	YES	NR	NR	YES	SLE NEPHRITIS	no	no	NO
292	M.S.	551509	YES	NO	NO	prerenal	3	HD	12	6	YES	YES	PR	CR	NO	NO	no	yes	YES
293	B.D.	550793	YES	NO	NO	renal	2	ND	9	0	NO	NO	PR	PR	NO	NO	no	no	NO
294	S.S.	566112	NO	YES	NO	renal	3	HD	19	9	NO	YES	NR	DEATH,27	YES	NO	no	no	NO
295	N.S.	550346	NO	YES	NO	renal	3	HD	21	12	NO	YES	PR	DEATH,29	YES	NO	no	no	NO
296	K.D.	196098	NO	NO	YES	renal	3	ND	9	0	YES	NO	PR	CR	NO	NO	yes	yes	YES
297	L.K.	571196	YES	NO	NO	renal	3	HD	13	13	NO	YES	DEATH,13	DEATH,13	NA	NO	no	yes	NO
298	A.S.	567577	NO	YES	NO	postrenal	2	ND	5	0	NO	NO	CR	CR	NO	NO	no	no	NO
299	L.K.	571694	YES	NO	NO	renal	2	ND	6	0	YES	NO	PR	CR	NO	NO	yes	yes	NO
300	B.S.	569696	YES	NO	NO	renal	2	ND	9	0	NO	NO	PR	PR	NO	NO	yes	no	NO
301	G.S.	588907	YES	NO	NO	prerenal	3	ND	7	0	NO	NO	CR	CR	NO	NO	yes	yes	NO
302	K.S.	591256	YES	NO	NO	prerenal	3	ND	7	0	YES	NO	PR	CR	NO	NO	yes	yes	NO
303	J.S.	122982	YES	NO	NO	prerenal	2	ND	8	2	NO	YES	DEATH,8	DEATH,8	NA	NO	yes	no	NO
304	B.K.	389696	YES	NO	NO	prerenal	2	ND	5	0	NO	NO	CR	CR	NO	NO	yes	yes	NO

S.no.	Name	MRD No.	Age	Gender	Rural/ Urban	Comorbidities	Comorbidity COAD	Comorbidity CLD	Comorbidity DM	Comorbidity HTN	Comorbidity CKD known	Comorbidity CKD (total)	Comorbidity CAD	Comorbidity HCV	Comorbidity HBV	Comorbidity HIV	Comorbidity MALIGNANCY	Comorbidity NEPHROLITHIASIS	Comorbidity TB	Comorbidity RA	Comorbidity CVA	Primary Etiology OF AKI	MULTIFACTORIAL AKI	ETIOLOGY GROUP
305	D.R.	591264	46	F	U	2	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	YES	DRUG
306	H.K.	582864	54	F	U	2	NO	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	YES	CARDIORENAL
307	H.K.	580424	65	F	U	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	CLL,TLS	NO	TLS
308	H.R.	591328	43	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
309	K.K.	579774	27	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREPUREAL SEPSIS/IUD	NO	SEPSIS
310	A.K.	586317	49	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
311	G.K.	591259	53	F	U	2	YES	NO	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
312	A.G.	591977	51	M	R	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- ARB	NO	DRUG
313	J.S.	588183	48	M	R	2	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
314	M.R.	580026	40	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
315	S.R.	591226	56	F	R	2	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	TROPICAL-DENGUE	NO	TROPICAL FEVER
316	S.L.	573110	57	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
317	A.K.	581419	46	F	R	1	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
318	K.D.	591293	32	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREECLAMPSIA/HELLP	NO	PREECLAMPSIA
319	B.K.	582135	40	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
320	S.D.	602505	43	M	U	1	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
321	S.A.	611659	51	F	R	2	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
322	K.D.	611858	57	F	R	2	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
323	S.S.	606031	48	M	R	1	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
324	N.K.	600852	51	F	U	1	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
325	S.S.	611575	49	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
326	M.B.	609804	34	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SNAKE BITE	NO	SNAKE BITE
327	A.S.	609080	38	M	R	2	NO	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
328	A.K.	609318	61	M	R	2	NO	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	YES	LVF/CARDIORENAL	NO	CARDIORENAL
329	B.K.	611482	62	F	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
330	A.S.	595927	63	M	R	2	NO	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
331	G.S.	400322	64	M	R	≥3	NO	YES	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	YES	SEPSIS
332	C.R.	610082	33	F	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PPH	NO	HYPOVOLEMIA
333	V.K.	609882	43	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
334	M.S.	606323	46	M	U	2	NO	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
335	M.S.	609368	47	M	U	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	NO	SEPSIS
336	A.D.S.	598614	49	F	U	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-SEPSIS/D.FOOT	NO	SEPSIS
337	S.D.	608551	40	F	U	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-ENTERIC	NO	TROPICAL FEVER
338	V. S.T.	611524	38	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-DENGUE	NO	TROPICAL FEVER
339	S.S.	610870	55	M	U	2	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	TROPICAL-DENGUE	NO	TROPICAL FEVER
340	S.L.B.	509079	62	M	R	2	YES	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	NO	CARDIORENAL
341	A.K.	622064	27	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-TRAUMA/RMYOLYSIS	NO	CRUSH INJURY
342	M.K.	618167	65	F	R	≥3	NO	NO	NO	YES	YES	YES	NO	NO	NO	NO	YES	NO	NO	NO	NO	SEPSIS/ CHOLENGITIS	NO	SEPSIS
343	P.K.	623461	39	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OPC POISONING	NO	POISONING
344	G.S.	626101	41	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
345	S.S.	626834	43	M	U	2	NO	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	NO	DRUG
346	B.K.	624157	47	F	R	1	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
347	K.D.	590357	48	F	R	1	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	ACUTE PANCREATITIS	NO	HYPOVOLEMIA
348	R.K.	626715	63	M	R	1	NO	NO	YES	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	YES	SEPSIS
349	R.A.	626072	62	F	R	2	NO	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	YES	CARDIORENAL
350	N.S.	617164	60	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
351	J.K.	595798	29	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREGNANCY-HUS	NO	HUS
352	M.K.	625829	31	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREPUREAL SEPSIS/IUD	NO	SEPSIS
353	R.S.	628777	40	M	R	2	NO	NO	YES	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	? DRUG- CAM	NO	DRUG
354	N.K.	626821	41	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-SCRUB	NO	TROPICAL FEVER
355	J.K.	628623	43	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/SEPTIC ARTHRITIS	NO	SEPSIS
356	A.P.S.	627936	49	M	R	1	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-PAPILLARY NECROSIS	NO	U.O.
357	R.V.	623245	48	F	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- ACEI	YES	DRUG
358	S.K.J.	630386	42	M	R	1	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
359	M.K.	629615	47	F	U	1	NO	NO	NO	YES	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	TROPICAL-LEPTO	NO	TROPICAL FEVER
360	M.S.	639191	51	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
361	S.S.	645072	51	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/GASTROENTERITIS	YES	SEPSIS
362	G.K.	645052	56	F	R	1	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/SBP	YES	SEPSIS
363	K.J.K.	646347	58	F	R	1	NO	NO	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	YES	SEPSIS
364	R.S.	643364	59	M	U	1	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	DRUG- NSAIDS	NO	DRUG
365	A.K.	646287	43	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
366	G.S.	646317	52	M	U	2	NO	NO	YES	NO	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
367	D.K.	644565	55	M	R	2	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PYELONEPHRITIS	YES	SEPSIS
368	S.R.	643392	22	F	U	0	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	OBS-APH	NO	HYPOVOLEMIA
369	B.R.	643875	40	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
370	R.K.	645735	54	M	U	2	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	YES	NO	NO	DRUGS-RIFAMPICIN	NO	DRUG
371	K.K.	646339	32	F	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	OBS-PREPUREAL SEPSIS	NO	SEPSIS
372	B.R.	648547	53	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	YES	SEPSIS
373	S.R.	649170	62	F	R	2	NO	NO	NO	YES	NO	YES	YES	NO	NO	NO	NO	NO	NO	NO	NO	LVF/CARDIORENAL	NO	CARDIORENAL
374	K.L.	649218	39	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	PARAQUOT POISONING	NO	POISONING
375	K.S.	649243	41	M	U	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SUR-NEPHROLITHIASIS	NO	U.O.
376	K.K.	654311	40	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
377	G.S.	641608	51	M	U	1	NO	YES	NO	NO	NO	NO	NO	YES	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA
378	B.S.	662433	56	M	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SNAKE BITE	NO	SNAKE BITE
379	J.S.	662462	57	M	R	1	YES	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	SEPSIS/PNEUMONIA	NO	SEPSIS
380	H.K.	660852	35	F	R	0	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	NO	A.G.E	NO	HYPOVOLEMIA

S.no.	Name	MRD No.	Medical AKI	Surgical AKI	Obstetric AKI	TYPE OF AKI	stage	Initial dialysis modality	Hospital stay	ICU Stay	HYPOTENSION	OLIGURIA	outcome discharge	Outcome 1 month	Readmission	Biopsy	Infective	Tropical etiology	Need of inotropes
305	D.R.	591264	YES	NO	NO	renal	2	ND	12	0	NO	NO	NR	NR	NO	AIN	no	yes	NO
306	H.K.	582864	YES	NO	NO	prerenal	3	ND	8	2	YES	NO	PR	PR	YES	NO	no	no	YES
307	H.K.	580424	YES	NO	NO	renal	2	ND	16	0	NO	YES	PR	CR	NO	NO	no	no	NO
308	H.R.	591328	YES	NO	NO	prerenal	3	CRRT	4	4	YES	NO	DEATH,4	DEATH,4	NA	NO	yes	yes	YES
309	K.K.	579774	NO	NO	YES	renal	2	ND	8	3	YES	NO	PR	CR	NO	NO	yes	yes	YES
310	A.K.	586317	YES	NO	NO	prerenal	2	ND	2	0	YES	NO	CR	CR	NO	NO	no	yes	NO
311	G.K.	591259	YES	NO	NO	renal	3	PD	5	5	YES	YES	DEATH,5	DEATH,5	NA	NO	yes	no	YES
312	A.G.	591977	YES	NO	NO	renal	2	ND	2	0	NO	NO	CR	CR	NO	NO	no	no	NO
313	J.S.	588183	YES	NO	NO	prerenal	3	SLED	21	7	YES	YES	DEATH,21	DEATH,21	NA	NO	yes	yes	YES
314	M.R.	580026	YES	NO	NO	prerenal	3	PD	7	5	YES	NO	DEATH,7	DEATH,7	NA	NO	yes	yes	YES
315	S.R.	591226	YES	NO	NO	prerenal	3	ND	9	3	NO	NO	PR	CR	YES	NO	yes	yes	NO
316	S.L.	573110	YES	NO	NO	prerenal	2	ND	7	0	NO	NO	PR	CR	NO	NO	yes	yes	NO
317	A.K.	581419	YES	NO	NO	renal	3	PD	15	8	YES	YES	NR	PR	YES	NO	yes	no	YES
318	K.D.	591293	NO	NO	YES	renal	3	HD	17	3	NO	YES	PR	CR	NO	NO	no	yes	NO
319	B.K.	582135	YES	NO	NO	prerenal	2	ND	7	0	NO	NO	PR	CR	NO	NO	yes	yes	NO
320	S.D.	602505	YES	NO	NO	prerenal	3	ND	13	8	NO	NO	PR	PR	NO	NO	yes	yes	NO
321	S.A.	611659	YES	NO	NO	prerenal	2	ND	9	0	YES	NO	CR	CR	NO	NO	yes	yes	NO
322	K.D.	611858	YES	NO	NO	renal	3	PD	6	6	YES	NO	DEATH,6	DEATH,6	NA	NO	yes	no	YES
323	S.S.	606031	NO	YES	NO	postrenal	3	ND	15	0	NO	NO	PR	PR	NO	NO	no	yes	NO
324	N.K.	600852	YES	NO	NO	renal	2	ND	10	3	YES	NO	PR	PR	NO	NO	yes	no	NO
325	S.S.	611575	YES	NO	NO	prerenal	3	CRRT	10	10	YES	YES	DEATH,10	DEATH,10	NA	NO	yes	yes	YES
326	M.B.	609804	YES	NO	NO	renal	3	HD	18	0	NO	YES	PR	PR	NO	ATN	no	yes	NO
327	A.S.	609080	YES	NO	NO	renal	3	SLED	4	2	YES	NO	DEATH,4	DEATH,4	NA	NO	yes	no	YES
328	A.K.	609318	YES	NO	NO	prerenal	3	ND	11	7	YES	NO	NR	PR	YES	NO	no	no	NO
329	B.K.	611482	YES	NO	NO	prerenal	2	ND	2	0	YES	NO	CR	CR	NO	NO	no	yes	NO
330	A.S.	595927	YES	NO	NO	renal	3	HD	9	0	YES	YES	PR	PR	YES	NO	yes	no	YES
331	G.S.	400322	YES	NO	NO	renal	2	ND	7	0	NO	NO	PR	PR	NO	NO	yes	no	NO
332	C.R.	610082	NO	NO	YES	prerenal	3	HD	20	6	YES	YES	NR	NR	YES	ACN	no	yes	NO
333	V.K.	609882	YES	NO	NO	prerenal	3	PD	4	4	YES	NO	DEATH,4	DEATH,4	NA	NO	yes	yes	YES
334	M.S.	606323	YES	NO	NO	prerenal	3	PD	19	10	YES	YES	NR	PR	YES	NO	yes	yes	NO
335	M.S.	609368	YES	NO	NO	renal	2	ND	8	0	NO	NO	PR	PR	NO	NO	yes	no	NO
336	A.D.S.	598614	NO	YES	NO	renal	2	ND	8	0	NO	NO	PR	CR	NO	NO	yes	no	NO
337	S.D.	608551	YES	NO	NO	prerenal	3	ND	10	0	NO	NO	PR	PR	YES	NO	yes	yes	NO
338	V. S.T.	611524	YES	NO	NO	prerenal	3	PD	3	3	YES	YES	DEATH,3	DEATH,3	NA	NO	yes	yes	YES
339	S.S.	610870	YES	NO	NO	prerenal	2	ND	6	0	YES	NO	CR	CR	NO	NO	yes	yes	NO
340	S.L.B.	509079	YES	NO	NO	prerenal	2	ND	11	5	YES	NO	PR	PR	NO	NO	no	no	YES
341	A.K.	622064	NO	YES	NO	renal	3	HD	20	13	YES	YES	NR	PR	NO	NO	no	no	YES
342	M.K.	618167	YES	NO	NO	renal	3	SLED	6	6	YES	NO	DEATH,6	DEATH,6	NA	NO	yes	no	YES
343	P.K.	623461	YES	NO	NO	prerenal	3	ND	26	6	NO	YES	DEATH,26	DEATH,26	NA	NO	no	yes	NO
344	G.S.	626101	YES	NO	NO	prerenal	2	ND	14	4	NO	NO	DEATH,14	DEATH,14	NA	NO	yes	yes	NO
345	S.S.	626834	YES	NO	NO	renal	2	ND	9	0	NO	NO	NR	NR	NO	NO	no	yes	NO
346	B.K.	624157	YES	NO	NO	prerenal	2	ND	3	0	YES	YES	PR	CR	NO	NO	no	yes	NO
347	K.D.	590357	YES	NO	NO	prerenal	3	ND	13	6	YES	YES	NR	PR	YES	NO	no	no	NO
348	R.K.	626715	YES	NO	NO	renal	3	ND	9	2	YES	NO	PR	PR	YES	NO	yes	no	YES
349	R.A.	626072	YES	NO	NO	prerenal	3	ND	11	2	YES	NO	PR	PR	YES	NO	no	no	NO
350	N.S.	617164	YES	NO	NO	prerenal	2	ND	7	0	NO	NO	PR	CR	NO	NO	yes	yes	NO
351	J.K.	595798	NO	NO	YES	renal	3	HD	18	3	NO	YES	NR	NR	YES	TMA	no	no	NO
352	M.K.	625829	NO	NO	YES	renal	3	PD	5	5	YES	YES	DEATH,5	DEATH,5	NA	NO	yes	yes	YES
353	R.S.	628777	YES	NO	NO	renal	2	ND	9	0	NO	NO	NR	NR	NO	AIN	no	yes	NO
354	N.K.	626821	YES	NO	NO	prerenal	3	ND	8	0	NO	NO	NR	PR	YES	NO	yes	yes	NO
355	J.K.	628623	YES	NO	NO	renal	2	ND	8	0	YES	NO	PR	CR	NO	NO	yes	no	YES
356	A.P.S.	627936	NO	YES	NO	postrenal	2	ND	11	0	NO	NO	PR	PR	NO	NO	no	no	NO
357	R.V.	623245	YES	NO	NO	renal	1	ND	3	0	NO	NO	CR	CR	NO	NO	no	no	NO
358	S.K.J.	630386	NO	YES	NO	postrenal	2	ND	6	0	NO	NO	CR	CR	NO	NO	no	yes	NO
359	M.K.	629615	YES	NO	NO	renal	3	SLED	15	5	YES	YES	DEATH,15	DEATH,15	NA	NO	yes	yes	YES
360	M.S.	639191	YES	NO	NO	renal	2	ND	9	0	YES	NO	PR	CR	NO	NO	yes	no	YES
361	S.S.	645072	YES	NO	NO	prerenal	2	ND	8	0	YES	NO	PR	CR	NO	NO	yes	no	YES
362	G.K.	645052	YES	NO	NO	prerenal	3	PD	4	4	YES	YES	DEATH,4	DEATH,4	NA	NO	yes	no	YES
363	K.J.K.	646347	YES	NO	NO	renal	3	SLED	11	5	YES	YES	PR	PR	YES	NO	yes	no	YES
364	R.S.	643364	YES	NO	NO	renal	2	ND	9	0	NO	NO	PR	PR	NO	NO	no	yes	NO
365	A.K.	646287	YES	NO	NO	prerenal	1	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
366	G.S.	646317	YES	NO	NO	renal	3	PD	7	7	YES	YES	DEATH,7	DEATH,7	NA	NO	yes	no	YES
367	D.K.	644565	YES	NO	NO	renal	2	ND	9	0	YES	NO	NR	NR	NO	NO	yes	no	YES
368	S.R.	643392	NO	NO	YES	prerenal	2	ND	5	0	YES	NO	CR	CR	NO	NO	no	yes	NO
369	B.R.	643875	YES	NO	NO	prerenal	2	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
370	R.K.	645735	YES	NO	NO	renal	3	ND	9	0	NO	NO	PR	PR	YES	NO	no	yes	NO
371	K.K.	646339	NO	NO	YES	renal	2	ND	10	0	NO	NO	NR	CR	NO	NO	yes	yes	NO
372	B.R.	648547	YES	NO	NO	renal	3	ND	8	0	YES	NO	PR	CR	NO	NO	yes	no	YES
373	S.R.	649170	YES	NO	NO	prerenal	2	ND	7	0	YES	NO	PR	PR	NO	NO	no	no	YES
374	K.L.	649218	YES	NO	NO	renal	3	HD	23	15	NO	YES	CR	CR	NO	NO	no	yes	NO
375	K.S.	649243	NO	YES	NO	postrenal	2	ND	8	0	NO	NO	PR	CR	NO	NO	no	yes	NO
376	K.K.	654311	YES	NO	NO	prerenal	2	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO
377	G.S.	641608	YES	NO	NO	prerenal	2	ND	5	0	YES	NO	CR	CR	NO	NO	no	yes	NO
378	B.S.	662433	YES	NO	NO	renal	3	SLED	23	13	YES	YES	NR	NR	YES	ATN	no	yes	YES
379	J.S.	662462	YES	NO	NO	renal	3	PD	18	15	YES	YES	NR	NR	YES	NO	yes	no	YES
380	H.K.	660852	YES	NO	NO	prerenal	1	ND	3	0	YES	NO	CR	CR	NO	NO	no	yes	NO

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No. BFUHS/2K18p-TH/ 2936 **Date** 19/3/19

The Principal,
DMC, Ludhiana.

Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 31.08.2018 has approved the plan of thesis of Sourabh Gupta, a student of your college for DM Nephrology along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Clinical profile and outcome of community acquired acute kidney injury

Supervisor
Dr. Preet Mohinder Singh
Asst. Prof.,
Dept. of Nephrology,
DMCH, Ludhiana.

Co-Supervisor
Dr. Vikas Makkar
Prof. & Head,
Dept. of Nephrology,
DMCH, Ludhiana.

The student has to submit the four copies of thesis, to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

SP Singh

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
2. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

CLINICAL PROFILE AND OUTCOME OF COMMUNITY ACQUIRED ACUTE KIDNEY INJURY

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN

PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DM

IN

NEPHROLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

FEBRUARY 2018

DR. SOURABH GUPTA

DEPARTMENT OF NEPHROLOGY
DAYANAND MEDICAL COLLEGE & HOSPITAL,
LUDHIANA

ABSTRACT OF PLAN OF THESIS

TITLE	CLINICAL PROFILE AND OUTCOME OF COMMUNITY ACQUIRED ACUTE KIDNEY INJURY
For Degree of	DM NEPHROLOGY
Candidate	**Dr. Sourabh Gupta**
Supervisor	**Dr. Preet Mohinder Singh,** Assistant Professor Department of Nephrology, Dayanand Medical College and Hospital, Ludhiana
Co- Supervisor	**Dr. Vikas Makkar,** Professor and Head, Department of Nephrology, Dayanand Medical College and Hospital, Ludhiana
Institution	Dayanand Medical College and Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: community acquired-AKI (CA-AKI) though well-known but is less studied and there is paucity of data in this setting.

Aims & Objectives: To identify the various etiologies and high risk groups of community acquired AKI and to evaluate the Renal outcome and mortality.

Material & Method: observational study will be done on Adult indoor patients who are diagnosed as Community acquired AKI based on KDIGO criteria.

Clinical Significance: To understand the profile of Community Acquired AKI in a low-middle income country of a tropical region to improve patient outcome.

Keywords: Community acquired AKI, KDIGO AKI criteria, Renal outcome, low-middle income country.

(Signature)	(Signature)	(Signature)	(Signature)
Candidate	Co-supervisor	Supervisor	Principal

CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"CLINICAL PROFILE AND OUTCOME OF COMMUNITY ACQUIRED ACUTE KIDNEY INJURY"** exist in the Department of Nephrology, Dayanand Medical College& Hospital, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis are genuine and is collected by the candidate himself under our supervision and guidance.

Supervisor

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Co-supervisor

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Date: 21/02/18

APPROVAL PROFORMA
BY
RESEARCH & ETHICAL COMMITTEE
DAYANAND MEDICAL COLLEGE AND HOSPITAL, LUDHIANA

Name of Candidate	Dr. Sourabh Gupta
Department	Department of Nephrology
Topic of Thesis	"CLINICAL PROFILE AND OUTCOME OF COMMUNITY ACQUIRED ACUTE KIDNEY INJURY"
Likely date of appearing in PG exam	May 2020
Date of enrolment	13th September 2017
Name of Head of Department	Dr. Vikas Makkar
Supervisor Co-supervisor	Dr. Preet Mohinder Singh Dr. Vikas Makkar
Signatures of Members of Research Committee with Stamp	**Signatures of Members of Ethical Committee with Stamp**
1. HOD Pharmacology	1. HOD Physiology
2. HOD Surgery	2. HOD Pathology
3. HOD Pathology	3. HOD Pharmacology
4. HOD Medicine	4. HOD SPM
5. Dean Academics	5. HOD Medicine
6. Vice Principal	6. HOD Surgery
	7. HOD Obst. &Gynae
	8. Legal Expert
	9. Secretary, IEC
	10. Dean Academics
	11.Vice Principal
	12 Principal

Approved: Yes/No

Chairperson,
Research Committee
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Research Committee

Approved: Yes/No

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Ethics Committee
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BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

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4	DATE OF ADMISSION OF COURSE	13 SEPTEMBER 2017
5	TITLE OF THE TOPIC	CLINICAL PROFILE AND OUTCOME OF COMMUNITY ACQUIRED ACUTE KIDNEY INJURY

6.0 BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

International Society of nephrology has recognised acute kidney injury as a major health concern and it aims to curb down mortality associated with acute kidney injury to zero by 2025 (0 by 25 initiative).[1]

Acute kidney injury (AKI), even when mild, is a well-known contributor to increased mortality and morbidity in hospitalized patients.[2] Hospital acquired-AKI (HA-AKI) has been the focus of research over the last two decades. A definition that maximally utilizes the available clinical information (serum creatinine and urine output) has been developed in this setting. The most recent definition for AKI has been organized by the Kidney Disease-Improving Global Outcomes (KDIGO) initiative which is derived mainly from the data of Hospital acquired-AKI (HA-AKI).[3] Community acquired-AKI (CA-AKI) though well-known for long but as an entity has gained increased attention over the last 2-3 years.

In global snapshot of AKI by ISN, majority patients had developed community acquired AKI (60%), and it was about 80% in Low & Low Middle Income Countries (LLMICs), even when rural areas were ill-represented from these countries. It shows the importance of Community acquired-AKI (CA-AKI).[1,4]

The disease burden in Low & Low Middle Income Countries (LLMICs) also the tropical countries is heavily influenced by local climatic and economic conditions. Thus, the spectrum of acute kidney injury (AKI) is fundamentally different to that described in affluent countries with temperate climates, where the disease burden is dominated by lifestyle related chronic diseases and degenerative disorders in

the elderly and AKI usually occurs as part of multiple organ involvement in an already hospitalized, elderly patient or after surgical or diagnostic interventions, and iatrogenic factors have an important role. Moreover, in contrast to the multi factorial origin of hospital acquired AKI, the aetiology of, community acquired AKI can usually be traced to a single cause, typically an infection, dehydration, exposure to an environmental toxin, envenomation by snakes or insects, or complications of out-of-hospital childbirth.[5]

These factors, associated with low levels of income, poor access to treatment, and social or cultural practices (such as the use of traditional herbal medicines and treatments) contribute to poor outcomes of patients with AKI.

Community-acquired AKI, though common, has been much less studied than Hospital acquired AKI. Because the causative factors of community-acquired AKI originate in the communities, environmental and lifestyle differences potentially affect the incidence and patterns of community. Therefore, uncovering the local features of community-acquired AKI is necessary for formulating site-specific preventive and therapeutic manoeuvres. Only Limited data are available on the aetiology and outcome of acute kidney injury in Indian population especially from this part of country, which creates a lacunae in our knowledge and need for this study.

6.2 REVIEW OF LITERATURE

CA-AKI is not a new concept. Some of the earliest case reports of AKI described persons with severe traumatic muscle injuries and subsequent rhabdomyolysis from the battlefield or areas of natural disaster.[6-8] Despite the preface community acquired, most of the data on this entity has been derived from hospitalized patients. Kaufman and colleagues performed the first formal analysis of CA-AKI.[9] They prospectively identified patients admitted to a single Veteran Affairs (VA) hospital over a 17 month period. The definition of AKI was derived from a schema for hospital acquired acute renal failure used by Hou et al.[10] They classified the majority of CA-AKI as pre-renal. Kaufman et al found that the prevalence of CA-AKI was 1% of hospital admissions, whereas HA-AKI was 4%. This is in contrast to later reports. For example, in the Liano et al study that explored the population level epidemiology of hospital-detected AKI (at or during admission), the major proportion of cases were identified on admission (60%).[11]

In studies that specifically examined the epidemiology of CA-AKI as compared to HA-AKI, CA-AKI has been found to be 2-3x more prevalent than HA-AKI. In these studies the cases of AKI were identified by international classification diagnostic (ICD) codes for ARF, thereby possibly underestimating the true prevalence of both CA- and HA-AKI in hospital.[12]

Population-wide incidence of CA-AKI was investigated by two separate groups. Liano and colleagues noted that of the total AKI identified in the hospital setting,

60.4% were identified on admission; however this study did not distinguish the incidence of CA-AKI from HA-AKI in their final analysis.[11]

Talabani and colleagues conducted the first population-wide analysis of changes in serum creatinine values specifically examining all variety of CA-AKI.[13] Of the patients who had creatinine measurements in an outpatient laboratory during a one month period, 0.7% were identified as meeting the criteria for CA-AKI (50% rise from baseline serum creatinine – which was measured in the 6 months prior to the index creatinine value. As compared to a random subset of the population who did not suffer CA-AKI, the CA-AKI individuals were older (70 v 57 yrs.) and more likely to have pre-existing CKD (37.8% v 27.7%, p=0.023).

Der Mesropian et al examined renal outcomes of patients with either CA- or HA-AKI as compared to patients who had no AKI.[14] Twelve percent and 6.1% of those with CA-AKI developed a doubling of serum creatinine or end stage renal disease at 3 years, respectively. These estimates were similar in patients with HA-AKI. Mortality was also very common among patients with any AKI and not different between the two groups. This was different from the findings of Wonnacott et al who looked at mortality at 14 months following CA-AKI. As compared to HA-AKI, patients with CA-AKI demonstrated a higher survival rate (55% v 36.9%, p<0.001).[15]

In an another study by Yafang Wang et al, a nationwide study on community acquired AKI, 4,136 patients were identified during the 2 month snapshots (January 2013 and July 2013). Of these, 2,020 (48.8%) had cases related to

decreased kidney perfusion; 1,111 (26.9%), to intrinsic kidney disease; and 499 (12.1%), to urinary tract obstruction.[16]

Notable differences exist between developing and developed countries in the incidence of CA-AKI, which seems higher in the former, but underreporting compounded by age and gender disparities makes available data unreliable.

Though reliable statistics on the patterns and prevalence of AKI in tropical countries are not available, but AKI is the most common renal emergency encountered.[17-20] Most published data are from single-centre studies conducted in urban areas and do not reflect the true prevalence of AKI, especially in rural areas.[21] Many rural patients who develop AKI do not reach urban hospitals. Furthermore, the available studies have used different denominators to calculate the incidence of AKI, making comparisons difficult. Thus, the reported incidence of AKI in tropical countries ranges from 0.31 cases per thousand hospital discharges [22] to 7.9 cases per thousand hospital admissions.[23]

Global snapshot of AKI by ISN highlighted the importance of community acquired AKI even in developed countries with 60% patients having community acquired AKI and it was about 80% in Low & Low Middle Income Countries (LLMICs). This was even when rural areas of developing countries were ill-represented as snapshot data mostly came from large urban centers.[3,4]

In a retrospective study done by Anupma Kaul et al in an urban centre of North India, where they excluded CKD and Diabetic patients, 2.5% patients with a mean age of 39.8 were diagnosed to have CA-AKI.[24] The most common cause of CA-AKI was medical (77.5%), followed by obstetrical (14.2%) and surgical

(8.3%) causes. Among the medical causes, acute diarrheal disease was the most common cause (29%), followed by malaria (18.8%) and sepsis (13.9%). Sepsis had the highest in-hospital mortality (46%). Nephrolithiasis was the most common surgical cause. Puerperal sepsis (44.1%), pre-eclampsia (23.5%), intrauterine death (11.8%), antenatal hemorrhage (11.8%) and post-partal hemorrhage (8.8%) were the obstetric causes.

In a study done by S. Goswami et al in an urban centre of North India, CA-AKI was more common than HA-AKI (57.2% vs 42.8).[25] There was no significant difference in age average and co morbidities between CAAKI and HAAKI. Dehydration and volume depletion were significantly more prevalent in patients with CAAKI (47.9% vs. 28.97% for HAAKI $p<0.0001$). While HAAKI was associated with a significantly higher prevalence of drug induced AKI (23.36% vs. 7% in CAAKI $p<0.0001$). The mortality in hospital was significantly higher in the HAAKI group compared to CAAKI group (51% versus 20% $p<0.0001$). This study highlights that risk factors for CAAKI and HAAKI are similar, with pre-existing CKD, diabetes, heart disease, hypertension, cancer and tuberculosis being common risk factors for both.

Despite our sophisticated understanding of the pathobiology of acute kidney injury, current prevention strategies are inadequate and current treatment options outside of renal replacement therapy are nonexistent.

International Society of nephrology has recognized acute kidney injury as a major health concern and it aims to curb down mortality associated with acute kidney injury to zero by 2025 (0 by 25 initiative) Knowledge of incidence and risk factors

is crucial because it drives local and international efforts on detection and treatment. Also, in developing countries, incidence varies seasonally; incidence peaks cause critical shortages in medical and nursing personnel.

Limited data are available on the etiology and outcome of Community acute kidney injury in Indian population. In this observational study, we plan to evaluate the clinical characteristics, etiologies, and outcomes of patients admitted to the hospital with community acquired acute kidney injury.

6.3 AIMS AND OBJECTIVES OF THE STUDY

To identify the various etiologies of Community acquired - AKI.

To identify high risk groups, who have increased risk of AKI.

To evaluate the Renal status at discharge and at 30 days post admission, 7 day and 30 day mortality, Need of ICU, Length of ICU stay and Total hospital stay and Readmission within 30 days of discharge.

7.0 MATERIAL AND METHODS

This is a prospective analysis of all patients who are admitted to the Dayanand Medical College and Hospital, Ludhiana, India from March 2018 to June 2019. It is a observational study and will include all patients during the study period who fulfill inclusion criteria.

7.1 SOURCE OF DATA: Indoor patients of Dayanand Medical College.

7.2 METHODS OF COLLECTION OF DATA:

INCLUSION CRITERIA

Patients ≥ 18 years admitted to Dayanand Medical College with Acute Kidney Injury at admission or within 48 hours of admission.

EXCLUSION CRITERIA

Age <18 years and >70 years

Referred following a hospital-acquired renal insufficiency

Consent refusal

CKD Stage 5 D

Particulars, detailed history and thorough clinical examination will be noted in a pre-structured proforma after written consent.

Defining CA-AKI

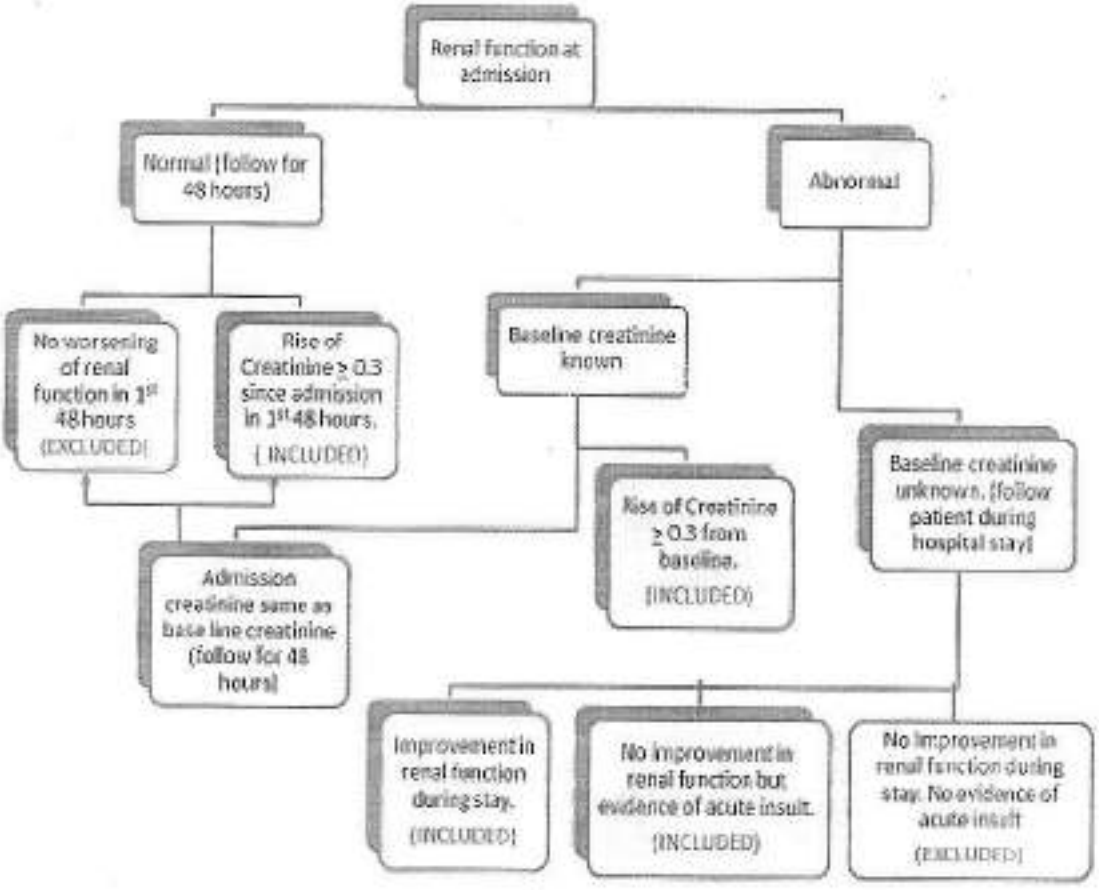
AKI will be defined as per KDIGO guidelines.

If the SCr value at admission or within 48 hrs of admission increase by ≥ 0.3 mg/dL from the baseline, It will be labelled as Community acquired acute kidney injury (CA-AKI).

Baseline SCr will be defined as the most recent measurement of SCr within the 3

months prior to admission.

For patient without baseline creat., an increased Scr level at admission and a trend of decreasing Scr levels during the hospital stay or evidence of acute insult in cases of non-resolution.


Renal function at admission
Normal (follow for 48 hours)
Abnormal
No worsening of renal function in 1st 48 hours (EXCLUDED)
Rise of Creatinine ≥ 0.3 since admission in 1st 48 hours. (INCLUDED)
Baseline creatinine known
Baseline creatinine unknown. (follow patient during hospital stay)
Admission creatinine same as base line creatinine (follow for 48 hours)
Rise of Creatinine ≥ 0.3 from baseline. (INCLUDED)
Improvement in renal function during stay. (INCLUDED)
No improvement in renal function but evidence of acute insult. (INCLUDED)
No improvement in renal function during stay. No evidence of acute insult (EXCLUDED)

OUTCOME:

Recovery of kidney function at discharge/follow up will be classified as

Full recovery- Serum creatinine level decreased to the threshold serum creatinine level, which was defined as ~ 10% of the baseline serum creatinine level.

Partial recovery- Serum creatinine level decreased by > 25% from the peak level, but remained above the threshold.

Failure to recover- the patient is dialysis dependent or scr level decreased by < 25% from the peak level.

Treatment withdrawal refers to patients with AKI who were recorded as severely ill but were discharged because they gave up further treatment in the hospital due to economic or personal reasons.

STATISTICAL ANALYSIS

A descriptive analysis will be performed. Continuous data will be presented as mean and standard deviation (m±Sd) and categorical data as a percent and 95% Confident Interval (CI). At the univariate analysis, proportions will be compared using a Pearson chi-squared test. Continuous data will be compared using t-test.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY

Yes, But as the study is observational Patients included in the study will only have to undergo investigations as per needed for patient management and no extra investigation will be done. For this patients included in the study will be informed regarding their inclusion and written informed consent will be taken for all the patients included in the study.

7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

Yes, Attached herewith

8. LIST OF REFERENCES

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PROFORMA

Sr.No.

Name Age

MRD No

Father/ Husband Name

Address Phone No.

Date of Admission Date of Discharge

Chief Complaints

Diagnosis

PAST HISTORY

DM	HTN	TB	CAD	COPD
CVA	HIV	HBV	HCV	TRAUMA
CKD	ANY OTHER			

FAMILY HISTORY

PERSONAL HISTORY

Smoking Alcohol Any other drug abuse

Signature of Candidate **Signature of Supervisor**

General Physical Examination

Blood pressure	Pulse	Respiratory Rate
SPO2	Temperature	JVP
Pallor	Icterus	Clubbing
Cyanosis	Edema	Lymphadenopathy

Systemic Examination

Respiratory System

CVS

CNS

Abdomen

Signature of Candidate **Signature of Supervisor**

Date				
Haematology				
Hb (g/dl)				
TLC(/µl)				
DLC				
Platelets (/µl)				
ESR				
Blood Biochemistry				
S.creatinine (mg/dl)				
B.urea (mg/dl)				
Sodium(meq/l)				
Potassium(meq/l)				
Calcium(mg/dl)				
Phosphorus(mg/dl)				
Albumin(g/dl)				
Uric Acid(mg/dl)				
Alk.Phosphatase(u/l)				
SGOT/PT(u/l)				
Total Bilirubin /Direct Bilirubin (mg/dl)				
Total Protein (g/dl)				
Blood Sugar - Random(mg/dl) - Fasting(mg/dl) - HbA1c(%)				
CPK (Wherever applicable)				
Urinary Sodium (Wherever applicable				
Urinalysis				

Signature of Candidate

Signature of Supervisor

Chest Xray Pa view

ECG

Anti HIV 1 and HIV 2

Hbsg

Anti-HCV

Ultrasound Abdomen (Where Ever applicable)

2-D Echo (Where Ever applicable)

Blood and Body Fluid C/S (Where Ever Applicable)

Date	Organisam	Senstivity

ANA (Wherever applicable)

ANCA (Wherever applicable)

C3 (Wherever applicable)

C4 (Wherever applicable)

ETIOLOGY OF AKI

1. **Dehydration**
2. **Tropical fever**
3. **Toxin – snake bite**
4. **NEPHROTOXIC DRUGS/ Alternative medication/ industrial-occupation exposure**

Name of drug	Date of Starting

Signature of Candidate **Signature of Supervisor**

5. **Obstructive uropathy**
6. **Obstetric causes**
7. **Sepsis**
8. **Any other**

KDIGO STAGE of AKI

TREATMENT MODALITY

Conservative Haemodialysis SLED CRRT PD

OUTCOME

Death Yes/No, if Yes, Date -

Cause-

Length of ICU stay

Length of Hospital stay

Creatinine: baseline- at discharge- 30 days post admission-

AKI Recovered

AKI Partial Recovered

AKI Not Recovered/Dialysis Dependent

READMISSION WITH IN 30 DAYS OF DISCHARGE

Date Diagnosis

Signature of Candidate **Signature of Supervisor**

INFORMED PATIENT CONSENT

Date: _____

I.....D/o, W/o, S/o..... am willing to participate in the study **"CLINICAL PROFILE AND OUTCOME OF COMMUNITY ACQUIRED ACUTE KIDNEY INJURY"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out of the study any time without assigning any reason.

....

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____ निवासी _____ इस शोधकार्य "CLINICAL PROFILE AND OUTCOME OF COMMUNITY ACQUIRED ACUTE KIDNEY INJURY" में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____ "CLINICAL PROFILE AND OUTCOME OF COMMUNITY ACQUIRED ACUTE KIDNEY INJURY" ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਤਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

_____ _____

ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

_____

ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

_____

ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ